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THE NASAL SEPTUM

PLASTIC REPAIR OF THE DEVIATED SEPTUM ASSOCIATED WITH A DEFLECTED TIP

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PHILADELPHIA

The importance of the nasal septum in man is self evident to the rhinologist. It divides the nasal cavity into two lateral passageways and thus forms the mesial wall of each nasal fossa. It also determines the line of the nasal dorsum.

In primitive races and in Caucasian children the nasal septum is usually straight and symmetric, but among Caucasian adults it frequently deviates. This deviation usually concerns the cartilage and the ethmoid portion, leaving the vomer normal or nearly so. Schaeffer [1] considered the perpendicular plate of the ethmoid bone (mesethmoid), which forms the cephalic third of the septum, of particular interest in septal deviation. The extent of the mesethmoid is variable, and the septal cartilage is correspondingly large or small.

During fetal life, infancy and early childhood, the nasal septum is usually symmetric and occupies the midline throughout its extent. Asymmetry has, however, been seen in early childhood, and marked deviation has been observed in some fetuses.

Deflection of the nasal septum in adults is so general as to be considered an almost normal condition. This deviation may vary from slight irregularities to those which entirely occlude one of the nasal fossae. In spite of these facts, many adults have essentially symmetric nasal septums.

Sheehan [2] pointed out that, regardless of the variations in the nasal framework which may account for its deformity, the first essential step is to return the nasal septum to its normal position. Since the septum separates the air passages and establishes the line of the dorsum, regardless of the degree of its deformity or weakening, it must be restored to fulfil these requirements before the other parts of the nasal framework can be adjusted effectively. The cartilage cannot be made straight without excision, and the amount of excision must vary according to the degree of the bend in the cartilage. Unless and until the septum has been set and straightened, nothing can be done toward assuring a correct line for the dorsum.

EMBRYONAL DEVELOPMENT OF THE NOSE

The human nose develops from anlages (nasal placodes) which appear about the fifth week of fetal life as thickenings of the ectoderm just above the oral plate of the embryo. The centers of these ectodermal thickenings become depressed by the rapid growth of the surrounding tissue, and in this way the early nasal pits are formed (fig. 1). For a short time the nasal pits open into the oral cavity. The primitive external nose is formed during the early development of the embryonic facial region. At the time of the fusion of the maxillary and the median nasal processes, the nasal pits become entirely separated from the oral fossa. A secondary connection with the roof of the primitive oral cavity appears in the 4 to 5 day embryo, due to thinning and final rupture of the bucconasal membranes, by which the primitive nares (choanae) are formed. Following this, the upper part of the oral cavity becomes continuous with the nasal cavity, owing to the formation of the palatal processes of the maxillas and the palatine bones. As these primary nasal fossae then grow backward by the resorption of the surrounding mesenchymal tissue, the definitive hard palate is fully developed and the nasal cavities become connected posteriorly with the pharynx by means of the consequently formed choanae (posterior nares). At this time they present the appearance of these cavities in the adult human being. The broad tissue mass between the nasal pits in the embryo becomes progressively narrower, to form the matured nasal septum,

Thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania, toward the requirements for the degree of Doctor of Medical Science (D.Sc.[Med.]) for graduate work in otolaryngology.

1. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

2. Sheehan, J. E.: Plastic Surgery of the Nose, ed. 2, New York, Paul B. Hoeber, Inc., 1936, p. 37.

which divides the nasal cavity into the paired lateral nasal fossae.[3]

In the fetus, on the posteroinferior part of the septum there are sometimes present from four to six oblique mucosal ridges (septal plicae). These folds usually disappear in early postnatal life.

POSTNATAL GROWTH OF THE NOSE

The later development of the nasal area has been reported by Rosenberger[4] from a study

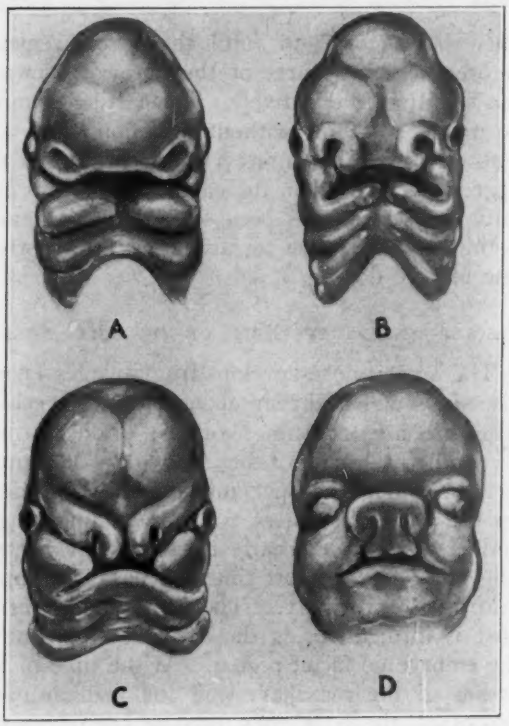

Fig. 1.—Development of the nasal septum from the primitive frontal prominence in the embryo: *A*, at 5 weeks (3.5 mm.); *B*, at 6 weeks (6.5 mm.); *C*, at 7 weeks (12 mm.); *D*, at 8 weeks (19 mm.). An original composite drawing from anatomic specimens and from Morris' Anatomy (Schaeffer[3a]).

pursued in age groups of from 3 months to 1 or 2 years. The younger group included the ages from 3 months to 5 years; the older group, those from 6 to 19 years. The observation was made that from infancy through adolescence the choanal and the facial growth, i. e., the entire nasal skeleton, drifts forward with growth of the cranium. By this process, the nasopharyngeal area is enlarged at an early period. By 5 years of age the pattern of upper facial development is established and no further changes in the growing nose take place in later childhood and adolescence. This fact would indicate that defects of growth originate in early childhood and persist throughout life.

ANATOMY OF THE NOSE

The nasal cavity (cavum nasi) consists of two irregularly shaped spaces. These spaces are the nasal fossae, which are separated by a median perpendicular septum (fig. 2 *A*). The anterior nares (piriform apertures) form the openings on the facial surface, and the posterior nares (choanae) constitute the openings into the pharynx.

The bony septum (septum nasi osseum) which divides the nasal cavity into two lateral passages is formed by the perpendicular plate of the ethmoid bone, the vomer, the rostrum of the sphenoid bone, the crest of the nasal bones, the frontal spine (frontal bones) and the medial crest formed by the meeting of the palatine processes of the maxillas and the horizontal portions of the palate bones. An angular notch on the anterior border is bounded by the perpendicular plate of the ethmoid bone and by the vomer. This notch is filled by the septal cartilage of the nose. The pharyngeal edge of the vomer, which divides the posterior nares, forms its posterior border.

Vomer.—The vomer (plowshare bone) (fig. 2 *B*) lies in the median plane and forms the dorsocaudal part of the nasal septum. It is an unpaired, thin, flat bone, irregularly quadrilateral in outline. There is usually a slight deflection to one side, which, however, only rarely involves the posterior margin.

The superior border or crest is expanded laterally into two alae (ala vomeris). The groove between the alae receives the rostrum of the sphenoid bone, and each ala comes into contact with the sphenoidal process of the palate and the process of the medial pterygoid plate. The inferior border is uneven and lies in the groove formed by the crests of the maxillary and palatine bones of the two sides. The anterior border slopes downward and forward and is grooved anteriorly for the septal cartilage of the nose. Posteriorly the vomer is united with the perpendicular plate of the ethmoid bone. The posterior border is smooth and rounded and

3. (*a*) Morris, H.: Morris' Human Anatomy, edited by J. P. Schaeffer, ed. 10, Philadelphia, Lea & Febiger, 1942. (*b*) Gray, H.: Anatomy of the Human Body, edited by W. H. Lewis, ed. 24, Philadelphia, The Blakiston Company, 1942. (*c*) Peter, K.: Die Entwickelung der Geruchsorganes, in Hertwig, O.: Handbuch der vergleichenden und experimentellen Entwicklungslehre der Werbeltiere, Jena, Gustav Fischer, 1902, vol. 2, p. 2. (*d*) Killian, G.: Zur Anatomie der Nase menschlichen Embryonen, Arch. f. Laryng. u. Rhin. **4**:1, 1896.

4. Rosenberger, H. C.: Growth and Development of Nasorespiratory Area in Childhood, Ann. Otol., Rhin. & Laryng. **43**:495 (June) 1934.

separates the posterior nares (choanae). The anterior and inferior borders approach each other at the anterior extremity of the bone, which presents a short margin behind the anterior prominence of the nasal crest of the maxillae. Near the anterior extremity, a small projection passes downward between the incisive foramens of the maxillas.[3a]

The ossification of the vomer takes origin from two bilaterally situated centers, which appear in the investing membrane of the ethmovomerine cartilage, at about the eighth embryonic week. The two lamellas thus formed unite below during the third month. Their union forms a shallow bony trough in which the cartilage lies. These lamellas grow upward and gradually fuse to form a rectangular plate of bone. The cartilage which lies between them then undergoes resorption. The alae which are on the superior margin and the groove in front indicate their original bilaminar development, although the adult vomer is not as a rule bilateral among mammals.[5]

Paired vomers are found in fish, and according to theory, the parts of the mammalian cranium which should correspond to the paired vomers were found in the palatine processes of the premaxillary structures. Paired vomers also occur in the embryos of some lower mammals, as the cat, the kangaroo and the opossum.[6]

Septal Cartilages.—The anterior part of the nasal septum is formed by the septal cartilage (cartilago septi nasi) (fig. 2 *C*). This cartilage is quadrilateral in shape and fits into the triangular space of the bony septum. There is a shallow groove on its anterosuperior margin which gradually narrows as the tip of the nose is approached, the superior borders of which are continuous with the lateral nasal cartilages; in their inferior two thirds these borders are separated by a narrow slit. The lower portion of this margin of the septal cartilage lies between the greater alar cartilages. Anteroinferiorly, the margin extends backward from the rounded anterior angle to the anterior nasal spine. Inferiorly it joins the medial crus of the greater alar cartilages and the mobile part of the nasal septum. The posterosuperior margin is attached to the perpendicular plate of the ethmoid bone, and the posteroinferior border is continuous with the vomer and the anterior portion of the nasal crest of the maxilla, where the cartilage is broader in a loose attachment to the nasal spine.

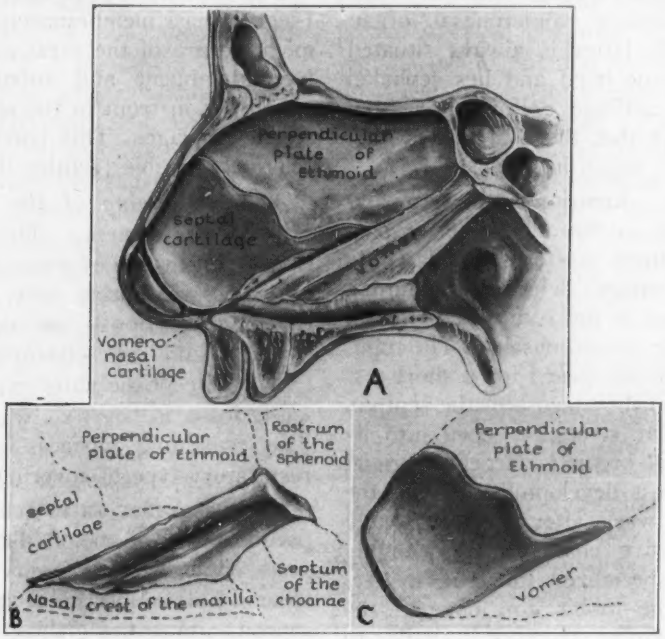

Fig. 2.—Anatomy of the normal nose.

The septal cartilage varies in size and shape according to the extent of the ossification of the bony septum. Posteriorly there is a variation in the septal cartilage which lies between the vomer and the ethmoid bone attachment, which forms the relation of the cartilage with the sphenoid bone (processus sphenoidalis septi cartilagini). This process may extend so far backward as to be in contact with the sphenoid body. This is seen particularly in children, in whom it may form a

5. Arey, L. B.: Developmental Anatomy, Philadelphia, W. B. Saunders Company, 1925. Morris.[3a] Gray.[3b]

6. Terry, R. J.: An Observation on the Development of the Mammalian Vomer, Anat. Rec. **3**:525, 1909.

horizontal ridge in one of the nasal cavities, giving rise to asymmetry of the nasal septum.

Vomeronasal Cartilages.—These cartilages (cartilagines vomeronasales, Jacobsoni) are two slender longitudinal strips, which lie along the anterior part of the inferior border of the septal cartilage, where they are attached to the vomer posteriorly, and to the maxilla and the septal cartilage anteriorly. The vomeronasal cartilages are sometimes not differentiated from the septal cartilages. Their greatest development in man occurs during the embryonic period. They are always well developed in animals, in which they form the surrounding medium of Jacobson's organ.[7] As regards man, on the contrary, it is to be doubted that these cartilages have any relation to the rudimentary vomeronasal organ (Jacobson) since the latter is always situated within the septal tissue itself and lies cephalad to the vomeronasal cartilages. For this reason, it should not be said that these cartilages support the vomeronasal organ in man.

Jacobson's Organ.—Anterior to the nasopalatine (incisive) canal, in the anteroinferior part of the nasal septum there is often seen bilaterally a small opening (ostium) which leads into a tubular sac. This sac is the vomeronasal organ of Jacobson (organon vomeronasale). The tubular formation extends backward for a short distance (2 to 6 cm.) in the mucosa of the septum. Many small glandular structures open into its lumen. In man this organ is largely vestigial and reaches its fullest development during the twentieth embryonic week, after which it usually undergoes a regressive change. It is highly developed, with a specialized function, in certain animals. In the rabbit, for instance, the mucosa lining the tubular structure is similar to the mucous lining of the nose.[8]

Jacobson's organ was the subject of extensive and careful study during the latter part of the nineteenth century. Outstanding work on the organ from the point of view of anatomy and histology was then done by Gratiolet[9] (1845), Kölliker (1877), Ganin[10] (1890), Anton[11] (1895) and others in the sheep, the pig, the rabbit, the guinea pig and the rat, as well as in birds, amphibia and many lower animal forms. More recently Frets[12] has reported on the histologic appearance and the nerve supply; Walter[13] has found that the passageway may open into the nasal cavity or that it may be closed off from the nose and open into the mouth. According to his study, this organ is most highly developed in snakes and lizards, is reduced in turtles and crocodiles, occurs embryonally in birds and mammals and is entirely absent in whales and bats. Pearlman[14] reported a particularly exhaustive study on the vomeronasal organ, which includes its gross anatomy, physiology and comparative anatomy.

The membranous portion of the nasal septum (septum nasi membranaceum) is formed by the medial crura of the great alar cartilages and by the integument and submucous tissue layer, which lies in front of the anterior border of the septal cartilage. This portion of the septum is relatively flexible (septum mobile nasi).

Mucosal Lining of the Nasal Cavity.—(*a*) Histologic Features: The walls of the nasal cavity are covered by a mucosal layer. There are two types of mucosa here, which represent the two nasal functions—one respiratory (pars respiratoria) and one olfactory (pars olfactoria). Only the cephalic third of the septum in each nasal fossa is covered by olfactory epithelium. The remaining surfaces of the septum bear respiratory type of covering.

The nasal mucosa in general is made up of a pseudostratified type of ciliated epithelium, which rests on a lamina of very vascular connective tissue. This connective tissue layer contains both mucous and serous glands. Of these glands, the small ones are convoluted, while the larger ones, which are more numerous, are of the tubular-acinous type and produce both mucous and serous secretions (schneiderian membrane).[15]

The ciliated cells extend through the entire thickness of the epithelial layer, with their slender terminals resting on the basement membrane. Irregular-shaped columnar or conical cells occupy the spaces between the stratified cells. These conical cells also rest on the basement membrane, but they do not reach the free

7. Jacobson, L.: Description anatomique d'un organ observé dans les mammiferes, Ann. d. Mus. d. hist. nat. **18**:1412, 1811.

8. Schaeffer,[1] p. 270. von Mikhailovics, V.: Anatomie der Nase, in Heymann, P.: Handbuch der Laryngologie und Rhinologie, Vienna, A. Hölder, 1900, vol. 3, no. 1.

9. Gratiolet, L. P.: Recherches sur l'organe de Jacobson, Thesis, Paris, 1845.

10. Ganin, M.: Thatsachen zur Frage über das Jacobsonschen Organ, Zoöl. Anz. **13**:285, 1890.

11. Anton, W.: Beiträge zur Kenntnis des Jacksónschens Organes der Erwachsenen, Ztschr. f. Heilk. **16**: 355, 1895.

12. Frets, G. P.: The Vomeronasal Organ, Morphol. Jahrb. **45**:566, 1912.

13. Walter, H. E.: Biology of Vertebrates, New York, The Macmillan Company, 1928.

14. Pearlman, S. J.: Jacobson's Organ: Its Anatomy, Gross, Microscopical and Comparative, with Some Observations on Its Function, Ann. Otol., Rhin. & Laryng. **43**:739 (Sept.) 1934.

15. Jordan, H. E.: A Textbook of Histology, New York, D. Appleton and Company, 1924.

surface. Mucus-containing goblet cells also rest on the basement membrane.

The basement membrane varies much in thickness. Though in general it is well developed, in some places it is hardly demonstrable. No capillaries appear within it, but since it is definitely cribriform, connective tissue fibers and leukocytes are able to pass through it.

The tunica propria is made up of a superficial fibroelastic layer, which is richly cellular, with occasional collections of lymphocytes. There are suggestions of discrete lymph nodules, though these are rare. Nearer the periosteum there is an abundant network of elastic fibers, which varies in density.[16]

The periosteum is only loosely attached to the underlying bone, while the attachment of the perichondrium is firm.[17]

The respiratory areas of the nasal mucous membrane are characterized by a covering of ciliated columnar epithelium. The cilia act to produce a current toward the pharynx.[18]

In the olfactory regions, the epithelium is columnar but without cilia, and there are none of the glandular elements which are present in the respiratory areas.

(*b*) Blood Supply: The arteries of the nasal mucosa arise from the sphenopalatine artery and its anastomoses with the ethmoidal, external nasal, septal and palatine arteries.

A network of veins goes both to the sphenopalatine vein and to the ethmoidal veins. There is also a rich venous communication with the veins of the dura and with the longitudinal sinus.

It is particularly of interest to the plastic surgeon that an examination of a large number of costal cartilages has revealed that their vascular system is precisely similar to that of the septal cartilages.[17]

(*c*) Innervation: The nerve supply of the nasal mucosa is through the olfactory nerve, the sphenopalatine ganglion, the nasal nerve and the internal branch of the ethmoidal nerve.

PATHOLOGY OF THE NASAL SEPTUM

The septum is subject to a variety of pathologic conditions. Perforation of the septum may be congenital. Schaeffer[8] found it in 2 of 300 or more specimens examined by him. This indicates the rarity of its occurrence. More frequently, however, it occurs in such diseases as syphilis and tuberculosis and such acute infections as diphtheria, scarlet fever or typhoid. Ballenger[19] considered the most common cause of this condition to be atrophic or perforating ulcer. Septal perforation may follow surgical operation and also frequent removal of crusts from the cartilaginous part of the septum with the fingers.

The septum may be the seat of acute infections, which may or may not go on to abscess formation.

Jacobson's organ, a glandular structure, rudimentary in man, which has already had consideration, occasionally undergoes pathologic changes. Brandt and Roper-Hall[20] have reported a cystic condition in the upper incisor region, at the junction of the maxilla with the floor of the nose, which arose from the embryonic epithelium of this organ. These authors emphasized that cysts in this location must be differentiated, since they may arise from other glandular structures present in the region. Cancerous new growth also may develop from Jacobson's organ. These growths are particularly malignant, characterized by their rapid and persistent infiltrative extension into the surrounding tissues.

Cade[21] observed that neoplasms of the nasal cavity which involve the septal cartilage do not respond well to treatment by radiation alone. Chondronecrosis is sure to follow, with severe and widespread septal infection. He recommends preoperative irradiation, then operative removal of as much as possible of the malignant tissue by means of diathermy needles, followed by further irradiation. If the growth is confined to the septum, both the septum and the columella should be removed to provide a cavity which can be satisfactorily irradiated. A later corrective operation assures little final deformity.

According to Charles,[22] it is generally true that a nose which lies more under the cranium than in front of it shows a greater tendency to disharmony of nasal form, one of the indications of which is a deflected septum.

This observation is of special interest in view of the fact that the most common abnormality of the septum, and the one with which this thesis is particularly concerned, is that of deviation, which

16. Hildebrand: Ueber das Verhalten des Epithels im Respiration Teil der Nasenschleimhaut, Jahrb. d. Hamb. Staatskronkenanst. **6**:25, 1897-1898. Morris.[3a] Gray.[3b]

17. Aymard, J. L.: Some New Points on the Anatomy of the Nasal Septum, J. Anat. **51**:293, 1917.

18. Lillie, H. D.: Some Practical Considerations of the Upper Respiratory Tract, J. Iowa State M. Soc. **13**:403, 1923.

19. Ballenger, W. L., and Ballenger, H. C.: Diseases of the Nose, Throat and Ear, ed. 8, Philadelphia, Lea & Febiger, 1943.

20. Brandt, W., and Roper-Hall, H. T.: Cysts of Jacobson's Organ, Brit. M. J. **1**:527 (March 3) 1940.

21. Cade, S.: Malignant Disease and Its Treatment by Radium, Baltimore, Williams & Wilkins Company, 1940, p. 476.

22. Charles, C. M.: The Cavum Nasi of the American Negro, Am. J. Phys. Anthropol. **14**:177, 1930.

may or may not be accompanied by deflection of the tip.[23]

Fractures and other accidental injuries of the nasal septum are frequent causes of deviation. These are particularly frequent in young children, in whom, according to Metzenbaum,[24] asymmetry of the nares is a positive diagnostic sign.

Fracture often causes dislocation of the anterior part of the nasal septum. The dislocated septal segment is pushed forward into the columella, which becomes broadened in this way, with narrowing of the nasal openings. This condition and its treatment have been well described by Cinelli.[25]

Cohen [26] emphasized the desirability of individual technic adapted to the requirements of each case, and in most instances if a plastic operation is indicated, this should be done at the same time as the correction of the septal deviation, or if these operations are not done at the same time, the plastic operation should precede the other by some months but should not follow it. He also warned against using dull forceps or force in the removal of fragments of the septal cartilage when operating.

Common regions of deflection are along the articulation of the vomer with the ethmoid plate. This articulation is particularly likely to be affected when the sphenoidal process of the septal cartilage extends backward for a considerable distance. At these articulations, angular deflections toward one or both sides may give rise to ridgelike folds and crests or to focal spurs. Although the septum itself may be essentially straight, a ridge or a spur on one side will produce the effect of asymmetry. The entire nasal septum may form a general convexity into one nasal fossa, with a corresponding concavity in the other; or the septum may deviate in an s-shaped manner and so encroach on both nasal fossae.

The reason for the occurrence of nontraumatic symmetry and deflection of the nasal septum remains largely theoretic. The condition is unknown in animals and savages. The degree of the facial projection from the axis of the skull (facial angle) is smallest in the ultracivilized races of man; it is considerably larger in the lower races and still larger in the anthropoid apes.

The nasal septum is apparently encroached on more and more from above by the forward extension accompanying the growth of the brain which occurs in the phylogenetic progress from the anthropoid apes to civilized man. If in this evolutionary process there is a balanced adjustment in the development of all parts of the cranium and face, this gradual change in the facial angle can hardly be a factor in septal asymmetry. If, on the other hand, all of these parts do not develop in such a harmonious manner, the changes resulting from the lessening of the facial angle may possibly cause encroachment on the nasal septum, surrounded as it is by other bony structures, and may cause it to be correspondingly deformed.[27]

Kyle [28] expressed the opinion that deformities of the nasal bones are often caused during labor. The position of the head in the birth canal permits considerable pressure on the soft, almost cartilaginous nasal bones.

My own observation on the displacement of the nasal septum during delivery testifies to the truth of Kyle's opinion. Metzenbaum [24] has stated that the contour of the nose of a child born by cesarean section is perfect, but in passing through the parturient canal, when the tip of the nose is depressed, the inferior border of the septal cartilage is sometimes forced out of the shallow groove it occupies in the floor of the nose, and the cartilage is unable to return to this groove spontaneously. The result is a displacement of the lower end of the septal cartilage, which then lies anteriorly across one naris.

The relation between rickets and asymmetry of the septum is implied by the frequency with which they are observed occurring together. This is also true of high-arched palate with nasopharyngeal obstruction and septal asymmetry. When a diagnosis of deflected tip associated with deviated septum is made, the technic for such correction is that here presented.

CORRECTION OF DEFLECTED TIP ASSOCIATED WITH DEVIATED SEPTUM

Preliminary Examination.—Each patient is given thorough individual study before operation. It is of particular importance to the success of the operation that the subject shall be in good physical condition. First, a thorough physical

23. Kopp, M. M.: Nasal Deformities Due to Septal Abnormalities, M. Rec. **151**:111, 1940.

24. Metzenbaum, M.: Dislocation of the Lower End of the Nasal Septal Cartilage, Arch. Otolaryng. **24**:78-88 (July) 1936.

25. Cinelli, A. A.: Alterations in Nasal Function Due to Anatomic Variation of the Nares, Arch. Otolaryng. **31**:53-64 (Jan.) 1940.

26. Cohen, S.: Role of the Septum in Surgery of the Nasal Contour, Arch. Otolaryng. **30**:12-20 (July) 1939.

27. Schaeffer, J. P., in The Human Face: A Symposium, Philadelphia, Dental Cosmos, 1935.

28. Kyle, D. B.: Textbook of Diseases of the Nose and Throat, ed. 5, Philadelphia, W. B. Saunders Company, 1914, p. 296.

examination is made. The results of the following special procedures are recorded:

1. Wassermann test
2. Complete blood count
3. Urinalysis
4. Determination of bleeding and coagulation time
5. Study of still photographs

If any of the laboratory tests indicate a pathologic condition, or if the physical examination reveals any disorder, operation is postponed until the condition of the patient is normal.

Collection of Instruments.—Instruments needed are a rake retractor, a recording syringe with

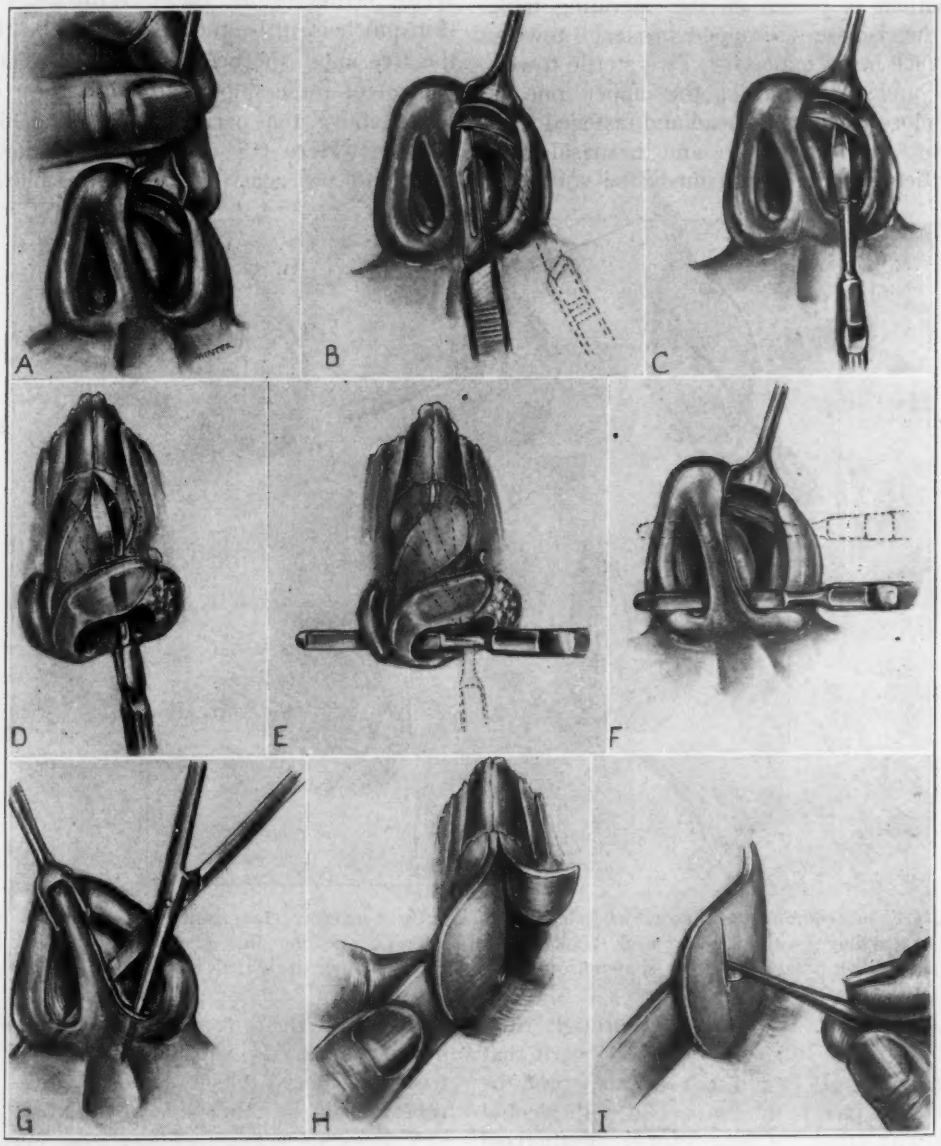

Fig. 3.—*A*, using the elevator to expose the deflected septum; locate the nasal fold. *B*, making incision. *C*, using Joseph's double-edged knife and retractor. *D*, undermining the skin over the upper lateral cartilage. *E*, using the button end knife in transfixing the nose. *F*, cutting down behind the columella. *G*, continuing the incision of *F* with scissors. *H*, bringing the septal cartilage into full view. *I*, making incision in the mucoperichondrium with the Ballenger knife.

(*a*) Direct profile
(*b*) Front face (erect) to show the degree of nasal deviation or other abnormality
(*c*) Front face with the head tilted back to show the nasal fossae through the nares

6. Profilometric measurement

various sized needles, a Bard-Parker knife (no. 11), a straight-pointed double-edged Joseph knife, a button end knife, a Ballenger knife, a straight fine-pointed scissors, a hemostatic forceps, a goose neck hook, a needle holder and suture material.

Preparation of the Operative Field.—Strict asepsis must be maintained. Four other principles outlined by Coates [29] should be particularly kept in mind:

1. Good light
2. Careful dissection
3. Bloodless field
4. Good anesthesia

The patient is placed on the operating table with the head closely wrapped in sterile towels, only the face being exposed. Two sterile towels are laid under the head; the upper one is wrapped closely about the head and fastened with towel clips. The entire face and the nasal cavities are then scrubbed with medicinal soft soap

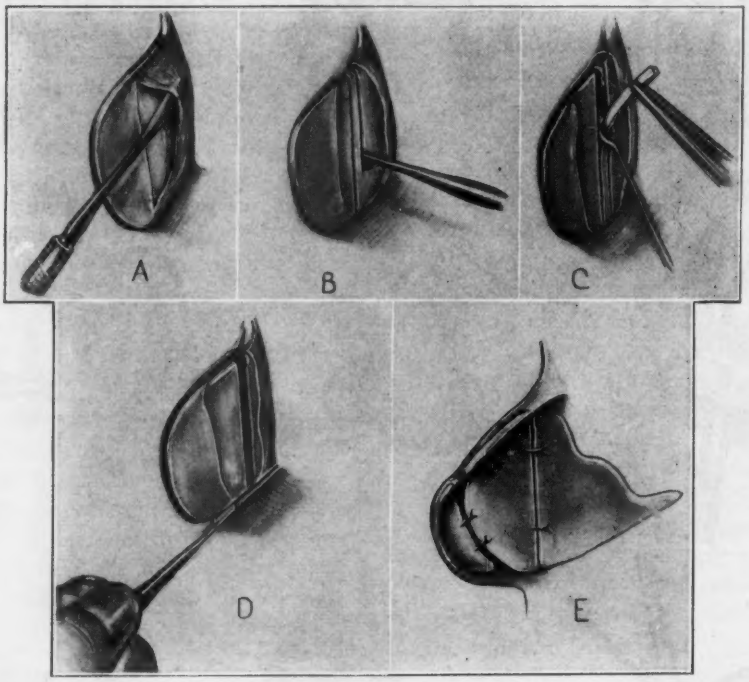

Fig. 4.—*A*, undermining the mucoperichondrium. *B*, cutting a narrow strip from the septum. *C*, removing the strip of cartilage with a goose neck hook and forceps. *D*, showing line of incision through the septal cartilage along the nasal floor. *E*, showing sutures in place holding straightened septum in position.

U.S.P. and water, followed by thorough rinsing with sterile water, with constant care that the nasal mucosa should not at any time be injured. The face is finally rubbed with alcohol and then with ether. The septum is anesthetized by means of cotton applicators wet with a solution of equal parts of 10 per cent cocaine hydrochloride and 1:1,000 epinephrine hydrochloride (or a 1 per cent solution of procaine hydrochloride to which 3 to 4 drops of 1:1,000 epinephrine hydrochloride have been added) placed inside the nasal cavity.

While this application is taking effect, the hairs about the nares are carefully removed with sharp-pointed scissors. After the removal of the cotton applicators, the nasal fossae with the exception of the vestibules are packed with long strips of half-inch sterile gauze, to prevent the escape of blood into the nasopharynx.

Anesthetization of the External Portion of the Nose.[29]—The nasal fold is located by elevating the ala of one side with a rake retractor and a 2 inch, 24 gage needle with syringe is placed at the center of the fold. With the overlying skin pinched up by the finger and the thumb of the free hand, the needle is advanced under the control of these fingers to the nasal root, in a plane above the perichondrium and the periosteum. Here 0.5 cc. of the solution in the needle (1 per cent procaine hydrochloride with 5 drops to the ounce of 1:1,000 epinephrine hydrochloride) is injected. This secures infratrochlear nerve block. The process is then repeated on the other side of the nose.

Then with a 1½ inch, 24 gage needle introduced intranasally at the extreme outer margin of the piriform opening and carried to a point which is determined by the intersection of (1) a vertical line drawn 1 cm. lateral to the inner canthus and (2) an oblique line from the columella to the outer canthus, 0.5 cc. of the solution is injected during the withdrawal of the needle. This secures infraorbital nerve block. The procedure is repeated on the opposite side.

29. Coates, G. M.: Lectures on Surgical Technique in Mastoid Operations, 1930.

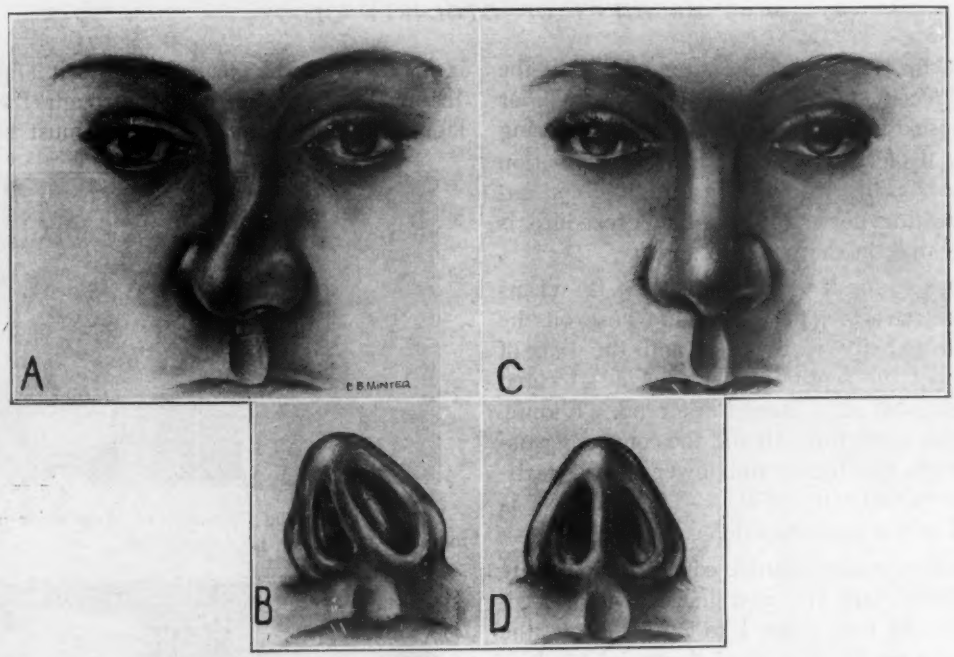

Fig. 5.—*A*, deformity due to deviation of the nasal septum. *B*, appearance of the septal buckle within the nares. *C*, deflected nose restored to normal contour. *D*, basal view for comparison with *B*.

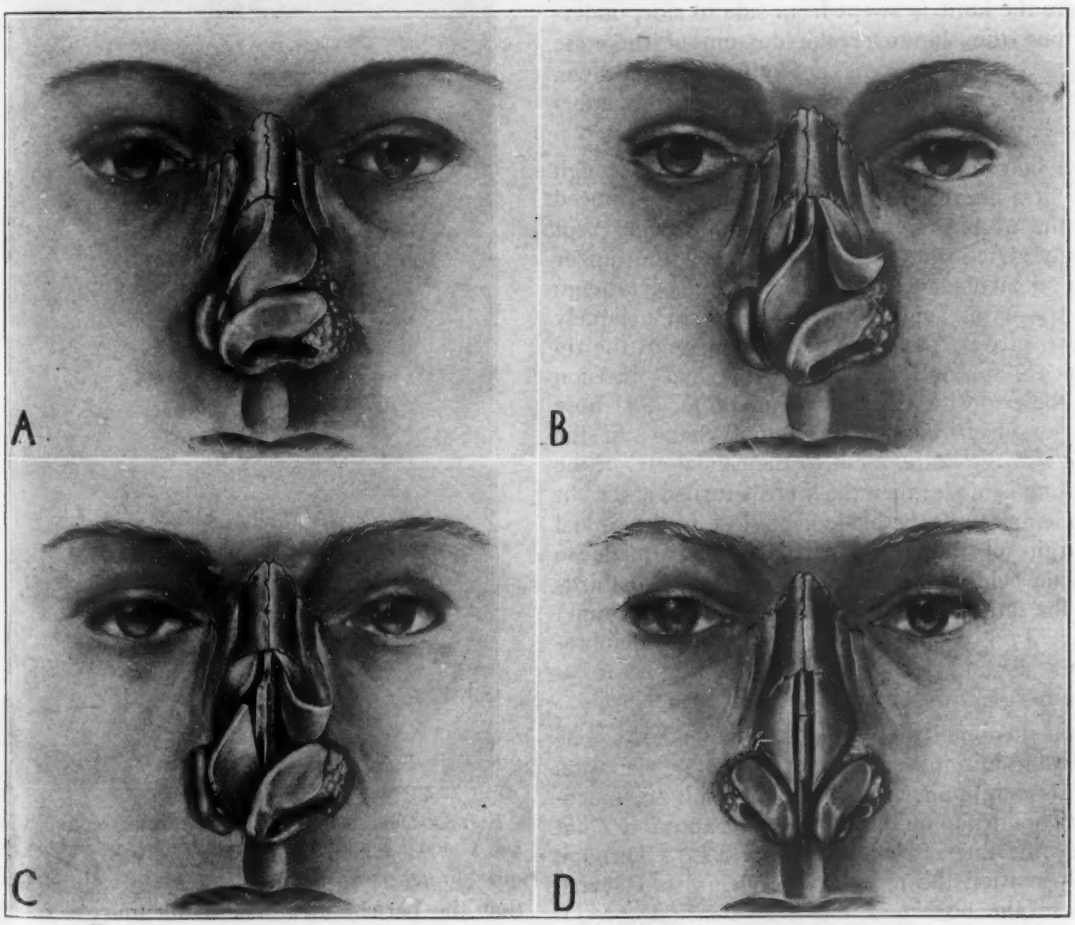

Fig. 6.—*A*, bony and cartilaginous structure of the deviated septum shown in figure 5 *A*. *B*, lateral cartilages separated from the deflected septum. *C*, septal cartilage after removal of the narrow strip and the upper lateral cartilage as shaped to conform to that on the opposite side. *D*, appearance of septal cartilage placed in midposition and sutured.

A 1 inch, 24 gage needle is introduced at the anterior nasal spine, advanced along the floor of the nose to the incisive foramen and during withdrawal of the needle 0.5 cc. of the solution is injected. This produces nasopalatine and anterior palatine nerve block. The procedure is then repeated on the opposite side.

Uncovering the Nasal Framework.—By means of a rake retractor, the ala of the nose on the side of convexity is elevated and the line of deviation of the septum (shelf; buckle) located (fig. 3 *A*). With a Bard-Parker no. 11 knife, the mucosa is cut through and the connecting tissue between the upper and lower lateral cartilages is incised (fig. 3 *B*). This incision is repeated on the opposite side.

A straight-pointed double-edged Joseph knife is introduced into the first incision (fig. 3 *C*) and is carried to a point 1 to 2 mm. above the caudal margin of the nasal bone in a plane immediately above the perichondrium of the upper lateral cartilage and underneath the skin. Here the knife is swept from side to side, undermining the skin over the dorsum of the nose (fig. 3 *D*) and then the knife is withdrawn. This procedure is repeated through the intercartilaginous incision on the right side.

A button end knife is then introduced through the first intercartilaginous incision and advanced to the nasofrontal suture, the knife being kept close above the perichondrium of the upper lateral cartilage and passed under the periosteum of the nasal bone. Then it is swept obliquely downward across the nasal dorsum until the tip appears through the intercartilaginous incision in the opposite vestibule (fig. 3 *E*). The nose is now transfixed, and without change of the plane of the knife, a cut to the caudal end of the septum is made; then the knife is turned at a right angle and with the blade kept close to the caudal margin of the septal cartilage, it is made to divide behind the columella through two thirds of the latter's extent (the membranous septum) (fig. 3 *F*). The section through the membranous septum is completed with straight sharp-pointed scissors (fig. 3 *G*).

At this point the septum can be pushed to one side and into full view (fig. 3 *H*). The operation on the septum itself is done by incising the mucoperichondrium on the convex side, anterior to but near the line of deflection (fig. 3 *I*). Through this incision the mucoperichondrium is elevated up to the buckle of the deflection (fig. 4 *A*). With a Freer or a Ballenger knife, an incision is made here, dividing the septal cartilage completely from the dorsum to the base. A second and parallel incision, exactly similar to the first, is made 0.1 to 0.5 cm. anterior to it (fig. 4 *B*). In making these incisions through the cartilaginous septum, particular care must be taken

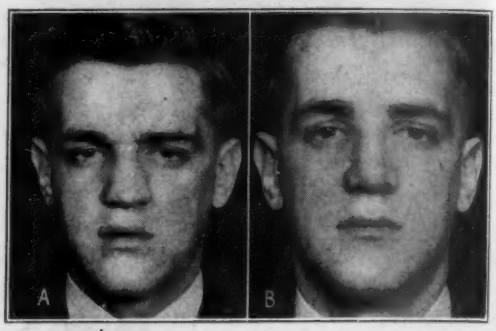

Fig. 7.—Depressed dorsum with deflected septum before (*A*) and after (*B*) correction.

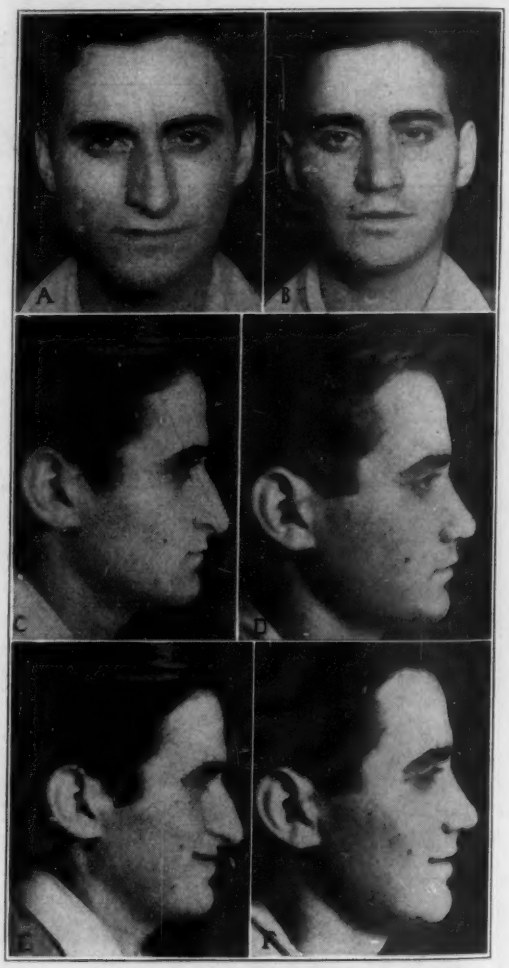

Fig. 8.—Deflected nose due to deflected septum before (*A*, *C* and *E*) and after correction (*B*, *D* and *F*).

that the perichondrium and the mucosa on the opposite side are not cut.[30] This point is worthy

30. Fomon, S.: The Surgery of Injury and Plastic Repair, Baltimore, Williams & Wilkins Company, 1939. Sheehan.[2]

of emphasis since this tissue carries the blood supply which is necessary to the life of the cartilage itself.

The narrow strip of cartilage between the two incisions is then removed by means of a goose neck (comma-shaped) hook. By inserting this hook over the upper end of the narrow fragment of cartilage (fig. 4 *C*), with the aid of forceps the fragment can be stripped entirely away from the perichondrium of the opposite side, and the buckle of the cartilage adjacent to it is thus removed.

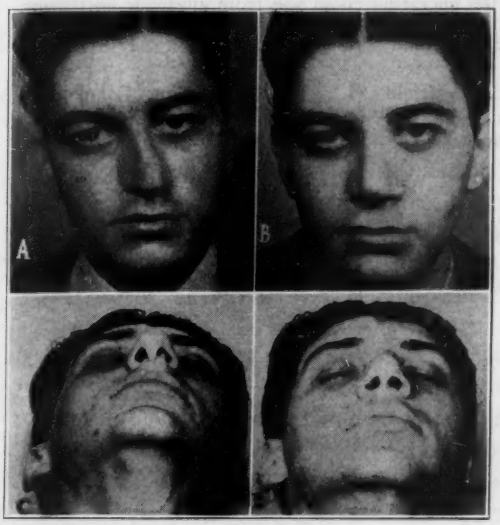

Fig. 9.—Deflected nose and septum before (*A* and *C*) and after correction (*B* and *D*).

Fig. 10.—Deflected nose and septum before (*A*) and after correction (*B*).

After the removal of the strip of septal cartilage, the base of the septum is freed by making an anteroposterior incision along the nasal floor through the cartilage on the operative side with a Freer or a Bard-Parker knife (fig. 4 *D*). Care must again be taken not to go through the underlying perichondrium.

The anterior segment of the septal cartilage being free, it is like a door without hinges and is so freely movable that it can be placed exactly in the central position. The surfaces of the carti-

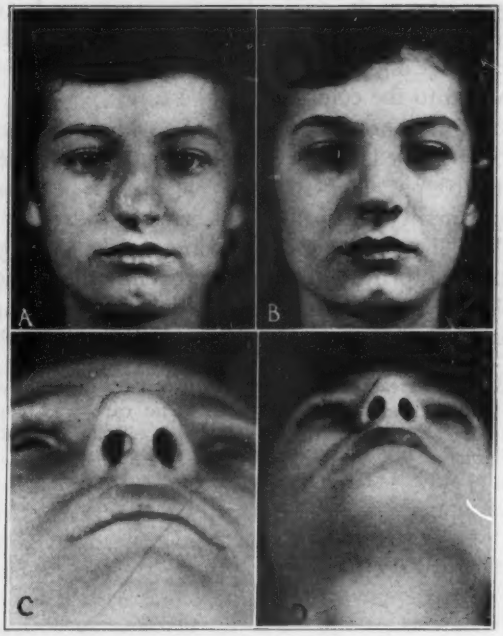

Fig. 11.—Deflected nose and septum before (*A* and *C*) and after correction (*B* and *D*).

Fig. 12.—Hump nose with deflected septum producing deflected tip before (*A* and *C*) and after correction (*B* and *D*).

Fig. 13.—Hump nose with deflected septum producing deflected tip before (*A*) and after correction (*B*).

lages where the strip has been removed are approximated and held together by two sutures. The front border of the anterior segment is then sutured to the columella (fig. 4 *E*).

With the nose in the desired position, an Asch tube wrapped in carbowax gauze impregnated with 10 per cent sulfathiazole and 2 per cent kephrine hydrochloride is inserted into each nostril.[31] These tubes check bleeding, afford bacteriostasis and provide drainage and ventilation. They hold the septum firmly in position and give support to the reconstructed tip. On the side of the former convexity a somewhat larger Asch tube is used than on the opposite side, in order to secure pressure on the septum. The tubes are left in place for four to five days. If desired, they may be removed and reinserted after cleaning.

An external adhesive dressing is finally applied, or a stent mold may be used following the Aufricht method.[32]

The original drawings were made from observation in the operating room and from anatomic specimens.

1332 North Franklin Street.

31. This treated gauze was furnished by the Johnson & Johnson research foundation. The carbowax compounds are polyethylene glycols of high molecular weight.

32. Aufricht, G.: Dental Molding Compound Cast and Adhesive Strapping in Rhinoplastic Surgical Procedure, Arch. Otolaryng. **32**:333-338 (Aug.) 1940.

CYSTADENOMA OF THE LARYNX

REPORT OF FOUR CASES

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Cystadenoma may be defined as adenoma that has undergone cystic degeneration. It is common in the breast and the ovary but extremely rare in the larynx. In fact, adenoma, from which it develops, is very unusual in this situation. A number of laryngologists of considerable experience never have observed a tumor with the microscopic diagnosis of simple adenoma of the larynx and some doubt its occurrence. However, numerous mucous glands are present in the larynx, and at times these may give rise to a glandular cell tumor. Recognition of the stage in the transition of an adenomatous tumor at which it has sufficiently pronounced cystic features to permit of its designation as cystadenoma will naturally depend on the personal equation of the pathologist. The present report is based on 4 cases in which advanced cyst formation was present.

HISTORICAL REVIEW

Few reports of cases of cystadenoma of the larynx appear in the literature. Casselberry [1] in 1891 removed from the arytenoid region a tumor which proved to be cystic, together with glandular tissue. Koschier [2] in 1897 described 2 cases in which such a neoplasm had produced prolapse of the ventricle of Morgagni. He recorded the gross and the histologic picture in detail and a method of surgical removal. Moore [3] in 1920 presented an excellent article on adenoma of the larynx in which he reviewed the 13 cases recorded in the literature up to that time and acknowledged that as a result of obstruction the tumor might become cystic and distended with mucin. Howarth and Chamings [4] in 1934 described a case in which two firm masses which appeared on indirect laryngoscopy to arise from the anterior commissure proved at the time of laryngofissure to be cystadenomatous tumors protruding from the ventricles. In 1938 New and Erich [5] in a study of 722 benign laryngeal tumors encountered at the Mayo Clinic included a single tumor diagnosed as adenoma of this structure. This tumor contained a large mucous cyst, and accordingly it is included in the present report. Jackson and Jackson [6] in their recent textbook stated that they had encountered 6 cases of laryngeal tumor in which the histologist found enough glandular-celled structure to justify calling the growth adenoma.

INCIDENCE

Since some of these cases have been reported as true cases of adenoma, the number of cases of cystadenoma actually recorded in the literature is difficult to state accurately. Probably fewer than 20 have been recorded but others unquestionably have been observed. Like laryngeal neoplasms in general, cystadenoma occurs more often among men than among women and in all of the reported cases the patient has been an adult. Three of our patients were male; 1 was female. However, 1 patient observed by us, a

From the Section on Laryngology, Oral and Plastic Surgery, Mayo Clinic.

Read at the meeting of the American Laryngological, Rhinological and Otological Society, Inc., New York, June 10, 1944.

1. Casselberry, W. E.: Cystoma of the Arytenoid Region of the Larynx, with the Report of a Case, J. A. M. A. **16**:338-341 (March 7) 1891.

2. Koschier, F.: Ueber Prolapsus sinus Morgagni, Wien. klin. Wchnschr. **10**:815-820 (Sept. 16) 1897.

3. Moore, I.: Adenomata (Glandular Tumors) of the Larynx, J. Laryng., Rhin. & Otol. **35**:65-75 (March) 1920.

4. Howarth, W., and Chamings, A. J.: Cystic Adenomata of the Ventricles of Morgagni, J. Laryng. & Otol. **49**:324-325 (May) 1934.

5. New, G. B., and Erich, J. B.: Benign Tumors of the Larynx: A Study of Seven Hundred and Twenty-Two Cases, Arch. Otolaryng. **28**:841-910 (Dec.) 1938.

6. Jackson, C., and Jackson, C. L.: Diseases and Injuries of the Larynx, New York, The Macmillan Company, 1942.

youth 17 years of age, had a tumor below the anterior portion of each vocal cord and had been hoarse since childhood. A tumor of the larynx had been removed from this patient at the age of 15 years. In all probability this tumor had been present during the first decade of life.

SITUATION

In view of the frequency with which certain benign tumors, especially papilloma, myxoma and cyst, occur in the larynx, the rarity of adenoma and accordingly of cystadenoma in this situation is striking. This is the more so when one considers that numerous mucous glands exist in most of the laryngeal mucosa.

In the majority of cases adenoma and cystadenoma of the larynx occur in the ventricles and ventricular bands, as is to be expected in view of the large number of mucous glands in these structures. The next most common sites of origin are about the epiglottis and in the subglottic region. Adenoma and cystadenoma may originate in any part of the larynx except possibly the free edges of the vocal cords, which are covered with squamous epithelium and are practically devoid of mucous glands. However, a tumor originating in the ventricle, the peripheral portion of a cord or the subglottic region may extend into the free edge of a vocal cord.

ETIOLOGY

Opinions concerning the mechanism of the formation of cystadenoma of the larynx differ. New and Erich stated that since adenoma, which is true neoplasm, has no ducts through which the contents of the acini can be expelled, it frequently becomes cystic. Koschier and Howarth and Chamings, on the other hand, ascribed the formation of cystadenoma to blocking of the ducts of the mucous glands, due to either acute or chronic inflammation with subsequent hyperplasia of the subepithelial connective tissue, followed by continued secretion of the glands into the acini and gradual distention of the latter to form cysts. Jackson and Jackson expressed the opinion that a form of cystic adenoma occurs in which there is primarily a retention of secretions with a secondary overgrowth of glandular and connective tissue.

PATHOLOGY

The definition of cystadenoma presumes that the tumor was simple adenoma primarily. As encountered in the larynx it shows great variation of the proportion of the cystic and adenomatous components, and a neoplasm that one pathologist would classify as adenoma might well be considered cystadenoma by another. The adenoma from which cystadenoma develops is described by Moore as simple glandular hyperplasia and is composed of glands held together by a relatively small quantity of loose connective tissue and covered with mucous membrane. This structure is retained in portions of the tumor as the cystic changes develop and gradually become the more prominent feature. A single cyst or multiple cysts of varying size may be present. They are lined with a single layer of low cylindric epithelium, which may be ciliated. The thickness of the walls varies greatly. The content of the cysts may be a clear mucinous fluid or a creamy or yellowish sebum-like material. The connective tissue surrounding the cysts varies in density and in amount. It is rather vascular and may contain serous spaces and muscle. There are frequent regions of inflammation with numerous lymphocytes, plasma cells and eosinophils. Adenomatous nodules may be present.

CLINICAL HISTORY

The history presented in cases of cystadenoma is that of a slowly progressive benign laryngeal neoplasm. Hoarseness is the outstanding feature and may be present for years. At times it dates from an acute inflammatory process in the respiratory tract, probably due to concurrent inflammatory enlargement of a tumor previously present. Cough is not a common complaint, but it may be paroxysmal and severe in cases in which the growth is pedunculated. In few of the recorded cases has dyspnea of appreciable degree been noted, but it may be pronounced, especially if an acute inflammatory process is present.

CLINICAL FINDINGS

Cystadenoma of the larynx presents a varying picture on clinical examination. Usually the tumor is sessile but it may be pedunculated. As a rule a single tumor is present, but there may be independent and almost symmetric growths on both sides of the larynx. The most frequent sites of origin are the ventricles, the ventricular bands, the epiglottis and the subglottic region. At times a bilateral tumor present in the subglottic region protrudes from the ventricles or produces a diffuse nodular or lobular enlargement of the ventricular bands. The tumor may range from a few millimeters to several centimeters in diameter. In a case observed by us, in which the cystadenoma was bilateral, the growth on the right measured 5.5 cm. in its greatest dimension and that on the left 4 cm. The cystic character of the growth may or may not be evident on clinical examination. A single large cyst may be present or the tumor may be multilocular. Commonly the growth is covered with normal-appearing mu-

cous membrane, but in some cases the mucous membrane is thickened and injected. The enlargement may be slightly yellowish in areas or throughout its extent, owing to the cystic contents showing through. Cases are reported in which the neoplasm involved the ventricle and produced prolapse of this structure by traction.

DIAGNOSIS

It is not likely that the laryngologist will be able to recognize a tumor of this type clinically. However, the long history, the involvement of the false cord or the ventricle with prolapse of the latter and the nodular or lobular character of the neoplasm would be suggestive of cystadenoma. Evidence of cystic change in a portion of the tumor, especially if this were yellowish, would be more strongly indicative of cystadenoma. However, a positive diagnosis is not probable prior to surgical removal and in most instances not before histologic study. The condition must be distinguished from (1) true prolapse of the ventricle or of the sacculus; (2) adenoma, the appearance of which is usually identical with that of its cystic counterpart; (3) myxomatous polyp; (4) the various granulomatous lesions and (5) adenocarcinoma or other malignant tumors, especially those originating in the ventricle.

TREATMENT

The treatment of a tumor of this type depends on its situation and size. A readily accessible growth of limited extent can be removed by indirect or direct laryngoscopy, but an extensive growth may require laryngofissure. Indirect laryngoscopy with removal of the tumor by means of a curved forceps and subsequent electrocoagulation is feasible but is gradually becoming a lost art in the treatment of benign laryngeal neoplasms generally. Direct laryngoscopy, with either the laryngoscope or the suspension apparatus being used, is preferable in most instances. Suspension laryngoscopy usually is the treatment of choice for the removal of the tumor since it affords a wider field of vision than other methods and permits the surgeon to use both hands, thus enabling him to carry out limited dissection. If the tumor extends deeply in the region of the ventricle or the ventricular band or if it is growing beyond the larynx, thyrotomy probably will be necessary.

Unless the tumor is obviously benign and well localized, with its limits definitely evident on direct examination, biopsy should be performed before treatment is instituted. The picture presented by cystadenoma of the larynx may be closely simulated by a malignant tumor developing in the ventricle.

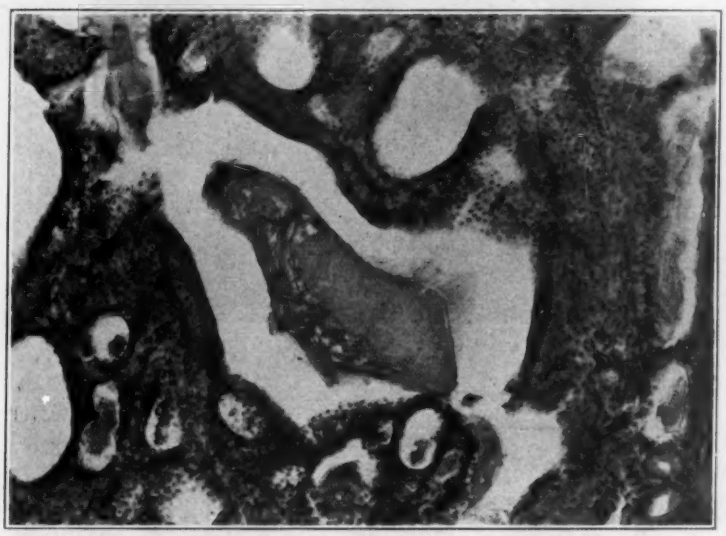

Fig. 1 (case 1).—Cystadenoma of the larynx; × 95. Multiple cysts of varying size are present. A number of these contain mucoid secretion.

In 3 cases in this group the tumor was removed with cutting laryngeal forceps and its attachment was electrocoagulated, suspension laryngoscopy being used. In the fourth case marked nodular enlargement of both ventricular bands was present and narrowed the airway sufficiently to produce considerable respiratory difficulty. This had been aggravated by a recent acute inflammatory process. Tracheotomy was necessary prior to investigation, laryngeal suspension being used. The fulness involving the anterior portion of the right side of the larynx appeared cystic, and a large cyst was opened when an attempt was made to secure a specimen of tissue for biopsy. Approximately 10 cc. of

thick, yellowish, purulent-appearing fluid escaped with collapse of a part of the mass on this side. On microscopic examination the specimen removed proved to be inflammatory tissue. The lesion was thought to be an extensive cyst, and exploration was performed through a thyrotomy

of multilocular cysts of some type. The tumor extended well above and lateral to the superior border of the thyroid cartilage on both sides. During the dissection rupture of some of the smaller cysts occurred with escape of sebum-like material, but the removal of both growths ap-

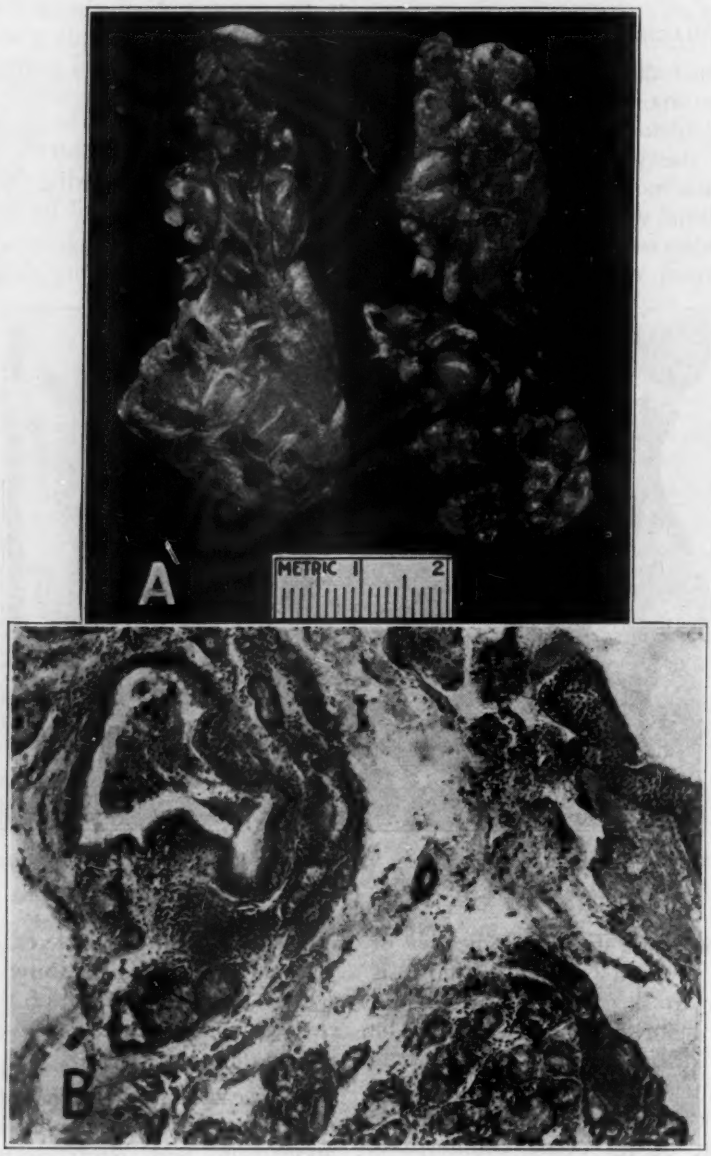

Fig. 2 (case 2).—*A*, tumors removed from the larynx, diagnosed microscopically as cystadenoma. The cystic portions of the tumors were distinctly yellowish. These tumors measured 5.5 by 2.5 by 1.3 cm. and 4 by 1.4 by 1 cm., respectively. *B*, section through one of the smaller cysts of one of the tumors shown in *A*; ×95. The mucoid content of the cyst contains numerous leukocytes, and the stroma surrounding the cyst shows marked leukocytic infiltration.

incision. An incision was made along the entire length of each ventricular band, and the mucosa was reflected. On both sides of the larynx this exposed a tumor consisting of multiple soft yellowish cystic sacs with firm fibrous tissue interspersed. Grossly the appearance was that

peared to be clean. The incisions in the mucosa were sutured after insertion of iodoform gauze packs, which were brought out through the thyrotomy incision. The growth on the right measured 5.5 by 2.5 by 1.3 cm.; that on the left, 4 by 1.4 by 1 cm. Microscopically each proved

to be very inflammatory cystadenoma. The patient's convalescence was uneventful.

RESULTS

The results in these cases have been entirely satisfactory to date as regards both eradication of the lesion and restoration of the voice. Since removal of the tumors, periods of five months, three and two-thirds years, four years, and seven and two-thirds years have elapsed, respectively, and there apparently has not been any return of the tumors. However, it should be borne in mind that neoplasms of this type are very slow growing, and a period of several years might be required for a recurrence to attain sufficient size to produce symptoms. In fact, in 1 of these 4 cases a tumor that may well have been of this nature had been removed from the larynx approximately twenty years prior to the patient's coming to us.

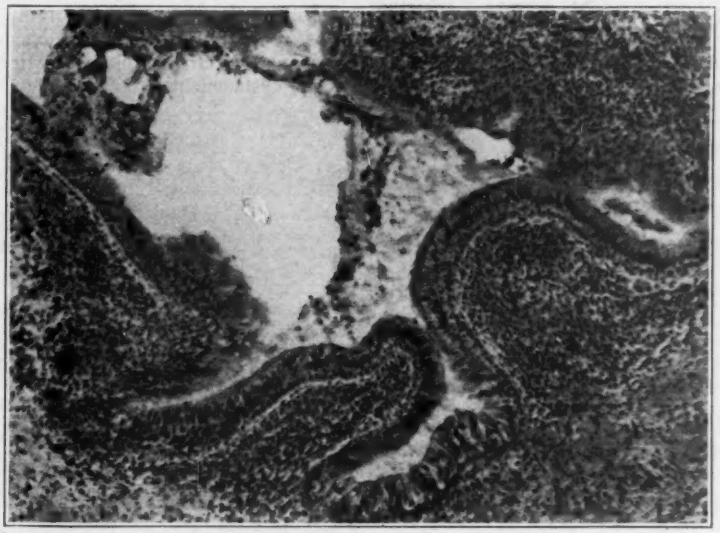

Fig. 3 (case 3).—Cystadenoma showing numerous mucous glands and a small cyst; × 60.

SUMMARY

Cystadenoma of the larynx is extremely rare. It apparently develops as a result of cystic degeneration of preexisting adenoma of this structure. The symptoms and clinical course are those of any very slowly progressive benign laryngeal tumor. Removal by means of suspension laryngoscopy is the most satisfactory form of treatment in most cases, although laryngofissure may be necessary because of the extent of the tumor. With complete removal the prognosis is good.

REPORT OF CASES

CASE 1.—A man 62 years of age came to the Mayo Clinic July 15, 1936 because of hoarseness of several months' duration. He had no other symptoms referable to the throat, and his general health was excellent. He had been examined by his local laryngologist, who found "growths" on the vocal cords and advised their removal.

Examination showed what was thought to be multiple polypoid degeneration of the larynx. On July 17, by suspension laryngoscopy, polypoid myxomatous tissue was removed from both vocal cords. A tumor about 1.5 cm. in diameter that extended into the glottis from underneath the anterior two thirds of the right vocal cord was grasped with forceps and excised with a laryngeal scalpel. Microscopically this tumor proved to be cystadenoma (fig. 1).

CASE 2.—A married woman 44 years of age came to the clinic May 16, 1939, complaining of hoarseness of nine months' duration. This had come on rather rapidly and had increased progressively. The patient also complained of some difficulty in breathing at night, which was relieved by coughing up mucus. She gave a history of having become hoarse after an attack of influenza twenty years previously and of having had a fibrous tumor 1.5 cm. in diameter removed from the left false cord. Radium had been applied subsequently.

The patient was a nervous, apprehensive woman who appeared to be in good general health except for marked hoarseness. A mass bulged the left ventricular band, and mucopus was present in the right nostril. There was clinical and roentgenographic evidence of pansinusitis. The nature of the laryngeal tumor was indeterminate clinically, and tracheotomy to be followed by suspension laryngoscopy and possibly laryngofissure was advised, but the patient refused treatment.

She returned one and a fourth years later (Aug. 2, 1940) because of increased hoarseness and rather severe respiratory difficulty. She had not received any treatment in the interim. She brought with her the tissue removed from the larynx twenty years previously, but this proved to be inflammatory. A mass still bulged the left ventricular band, but an almost symmetric, although somewhat larger and cystic-appearing tumor now involved the right false cord also.

On August 3 tracheotomy, suspension laryngoscopy and biopsy were performed. During the examination the tumor on the right ruptured and approximately

10 cc. of purulent-appearing material escaped. The tumor became flaccid. The pathologist reported the biopsy specimen to be inflammatory tissue.

The lesion was thought to be a large laryngeal cyst, and laryngofissure was done three and a half weeks later. When the mucous membrane of the ventricular bands was dissected back, a discrete tumor was exposed on each side. The tumors were composed of multiple yellowish cysts ranging up to a little more than 2 cm. in diameter, with small solid fibrous masses interspersed, these components being bound together with fibrous tissue and the whole having a rather definite capsule. The tumors extended well above and lateral to the superior border of the thyroid cartilage on each side. What looked like thin sebaceous material escaped from some of the ruptured loculi during removal, but the tumors appeared to have been taken out cleanly. Iodoform gauze packs were placed in the wounds, and the mucous membrane was sutured.

The tumors measured (right) 5.5 by 2.5 by 1.3 cm. and (left) 4 by 1.4 by 1 cm., respectively (fig. 2 *A*). On microscopic examination they were reported to be very inflammatory cystadenoma (fig. 2 *B*). The patient's convalescence was uneventful, and as far as is known she has not had any further trouble in this connection.

CASE 3.—A youth 17 years of age came to the clinic for examination on April 4, 1940 because of hoarseness dating from childhood. Two years previously an otolaryngologist had found a nodule on one of the vocal cords, and treatment with rest of the voice and intranasal applications had been carried out.

Our examination revealed yellowish tumors underneath the anterior thirds of both vocal cords, that on the left side being slightly larger than that on the right. Under suspension laryngoscopy the tumors were removed with cutting forceps. They proved to be cystic, that on the left measuring 5 mm. in diameter, that on the right 3 mm. Microscopically each proved to be cystadenoma (fig. 3). Recovery was uneventful. The patient has not returned to the clinic since.

CASE 4.—A salesman 56 years of age came to the clinic on Oct. 25, 1943 because of intermittent hoarseness of three years' duration. This had been constant for the past year. The patient had a chronic cough, and a number of years previously he had been thought to have pulmonary tuberculosis, although the diagnosis was not confirmed. Examination revealed fulness involving the anterior portion of the left false cord. On Nov. 1, 1943, with the patient under anesthesia induced by intravenous injection of pentothal sodium, a cystic mass measuring 1.5 by 1 cm. attached to the ventral aspect of the anterior half of the left ventricular band was removed under suspension laryngoscopy, and its base was electrocoagulated. Microscopically it proved to be cystadenoma.

SURGICAL TREATMENT OF ATROPHIC RHINITIS

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Atrophic rhinitis is degenerative rhinitis in which the normal ciliated columnar type of epithelium is replaced by a nonciliated, stratified squamous type. The mucus-secreting glands are greatly diminished in number but not totally absent, so that mucus is still secreted. Textbooks of otolaryngology describe two groups of cases: (1) cases in which no odor is present in the nose and (2) cases in which there is a fetid odor. The condition in the latter group is commonly called ozena.

CLINICAL CLASSIFICATION OF CASES OF ATROPHIC RHINITIS

From a clinical point of view atrophic rhinitis should be considered as of four types:

Latent Atrophic Rhinitis.—This type is characterized merely by a diminution in size of one or more turbinates and may be asymptomatic, requiring no treatment.

Simple Mild Atrophic Rhinitis.—This type is characterized by atrophy of either the middle or the inferior turbinate or both, on one or both sides, but no crusting is observed. This form usually occurs in old age and also may be asymptomatic, requiring no therapy.

Moderate Atrophic Rhinitis in Which Marked Crusting Occurs in Addition to Atrophy of the Turbinates.—This form may be called ozena if a stench is present. Hoople and Rowe [1] stated that "crusting did not appear without odor, nor odor without crusting. They went hand in hand in the history and in the treatment. Sixty per cent of patients gave a history of purulent discharge previous to the appearance of crusting and fetor." On examination of the nose of such a patient, the nasal chambers will be seen to contain large quantities of thick, greenish yellow or blackish crusts, which in patients who have neglected sanitary cleansing, almost completely obliterate the nasal cavities. On removal of these crusts, one is amazed at the spaciousness of the olfactory cleft. The middle and inferior turbinates are shrunken, owing to atrophy of the mucous membrane. Septal perforations resulting from the degenerate ulcers are not uncommon. The whole of the upper respiratory tract may become involved in this degenerative condition.

Fox, Dinolt and Morwitz [2] considered ozena as of two types: the primary or atrophic fetid rhinitis, and the secondary, which follows a chronic suppurative infection of the paranasal sinuses. These authors reviewed the literature and listed the causes of the primary type.

The symptom complex of atrophic rhinitis per se varies according to whether or not fetor is present and according to the degree of atrophy. If the bad odor is absent, the condition may be asymptomatic, discovered during a routine nasal examination. Other patients complain of weakness, lassitude, recurrent headaches and nasal obstruction. Highly sensitive patients may become melancholic or show introvert tendencies when they become aware of their obnoxiousness. The physician may make the diagnosis as soon as the patient enters his consulting room.

Present day medical therapy consists mainly of sanitary nasal douches in addition to administration of an estrogen. The estrogen may be applied topically in the form of a nasal spray or a pack, implanted subcutaneously or taken by mouth. Although there is no doubt concerning the relief patients will receive from treatment, the therapy still falls short of being satisfactory, and I believe surgical treatment is a method which offers hope for something more than transient relief.

Severe Atrophic Rhinitis or Terminal Ozena.—This type may develop in cases of long-standing ozena. The clinical description is exactly that of a nose destroyed by syphilis. However, no history of syphilitic contact or infection and no positive serologic test can be obtained. The nose is contracted and pulled inwardly, with loss of the cartilaginous septum and partial or complete loss of the bony septum. The ciliated epithelium is destroyed and replaced by scar tissue.

From the Department of Otolaryngology, New York Post-Graduate Medical School and Hospital, Columbia University.

1. Hoople, G. D., and Rowe, A. W.: Atrophic Rhinitis, Ann. Otol., Rhin. & Laryng. **36**:144, 1927.

2. Fox, N.; Dinolt, R., and Morwitz, S. M.: Atrophic Rhinitis with Ozena: Etiologic Factors, Arch. Otolaryng. **33**:1033 (June) 1941.

SURGICAL THERAPY OF MODERATE ATROPHIC RHINITIS

From the surgical point of view, cases of moderate atrophic rhinitis should be classified as follows: (1) cases in which the condition is unilateral and (2) cases in which it is bilateral.

Until the present, all surgical technics have employed the same principle, i. e., to narrow the nose. It has been definitely observed that a diminution of the spaciousness of the nasal chambers results in both subjective and objective improvement.

Since there are an absence of nasal cilia, a diminution in the secretion of mucus and a passing of large quantities of air through the olfactory cleft, the nose feels dry and the patient refrains from blowing it. However, some mucous glands still function, and the mucus that is secreted dries rapidly in the large amount of air entering the nose. Crusts form, which harden and cannot be dislodged by mere expiratory pressure. If the interior of the nose is narrowed surgically in addition to narrowing the alar openings, the quantity of air entering the nose is decreased so that the mucus remains catarrhal in character for a longer period and may be more easily evacuated by the patient.

The first attempt at narrowing the nose was at the end of the nineteenth century when Sanger [3] used metal obturators in the nose. Then Gersung [4] injected paraffin under the mucosa, which started the era of transplants. Lautenschläger [5] described his operation to narrow the interior of the nose by fracturing the nasoantral wall medially by the transantral approach. Hinsberg, Halle and Wachsberger [6] then modified Lautenschläger's operation. Kemler [7] used ivory implants on the floor of the nose and claimed that "all the ivory introduced into the septum was extruded after a shorter or longer period." Kasnetz [8] reported 6 cases in which ivory was implanted in the septum, with instant relief. Moreira [9] also modified Lautenschläger's operation and reported 48 per cent excellent results, 29 per cent good results, 21 per cent fair results and 2 per cent failures.

Bilateral Moderate Atrophic Rhinitis.—In the type of operation I advocate, only preserved septal cartilage is employed. The cartilage has been obtained from patients on whom submucous resection has been performed, and is thus free of perichondrium. The cartilage is merely washed in sterile saline solution and stored in 70 per cent alcohol (fig. 1). The principle of this operation, as in all the previous ones, is to diminish the air space in the nose in addition to diminishing the quantity of air entering the nose.

The crusts are first thoroughly removed by irrigation with a solution of sodium bicarbonate. Any adherent crusts are removed by a tight dry cotton tampon. The nose is then filled with a

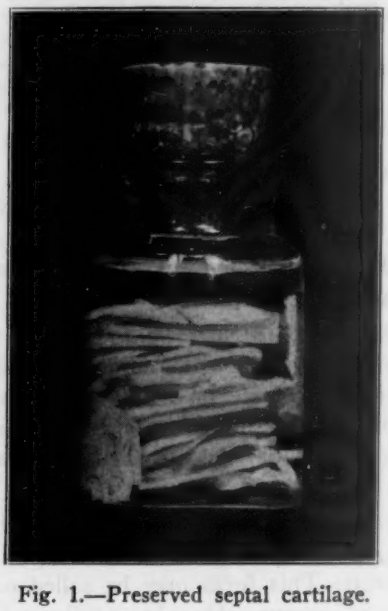

Fig. 1.—Preserved septal cartilage.

solution containing equal parts of a 10 per cent solution of a cocaine salt and 1 : 1,000 solution of epinephrine hydrochloride, which is allowed to remain in situ for ten minutes. A 1 per cent solution of procaine hydrochloride with eight drops of a solution of epinephrine hydrochloride to the ounce is infiltrated into the whole of the septum between the cartilage and the perichondrium. Infiltration causes additional anesthesia, hemostasis and ballooning of the mucosa. Dissection is thus made easier since the flaps are thickened and the tendency toward buttonholing is diminished. The columella and nasophiltral angle are also injected for hemostasis and anesthesia since small branches of the infraorbital nerve supply the columella and would not be blocked by the previous procedure alone. This measure also tends to alleviate the discomfort caused by pressure from the nasal speculum.

3. Sanger, M.: Ozenatherapie, Therap. Monatschr. **8**:502, 1894.

4. Gersung, R.: Ztschr. f. Heilk. **21**:199, 1900; cited by Kopp.[11]

5. Lautenschläger, A.: Arch. f. Laryng. u. Rhin. **31**:103, 1918; Grundlagen der Ozänatherapie, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **120**:7, 1929.

6. Wachsberger, A.: New Technic in Surgical Treatment of Ozena, Arch. Otolaryng. **19**:370 (March) 1934.

7. Kemler, J. I.: Implantation of Ivory in Ozena: Approved Technic; Further Observation, Arch. Otolaryng. **13**:726 (May) 1931.

8. Kasnetz, J. P.: Modified Technic of Ivory Implantation for Ozena, Arch. Otolaryng. **29**:699 (April) 1939.

9. Moreira, E.: Ozena (rinite atrófica fétida), Rev. brasil. de oto-rino-laring. **10**:5, 1942.

The initial incision is made in the vestibular skin at the anterior free margin of the septum, in either the right or the left nostril, and is just deep enough to expose the septum but not so deep as to go through the other side. The incision should extend from the highest part of the septum down to the anterior nasal spine. By means of a Freer elevator, the mucoperichondrial and mucoperiosteal flaps are elevated as completely as possible on both sides of the septum. The columella is now completely separated from the anterior free end of the septum. This should be performed only after the mucoperichondrium has been bilaterally elevated, since the septum will have a tendency to buckle when one is attempting blunt dissection.

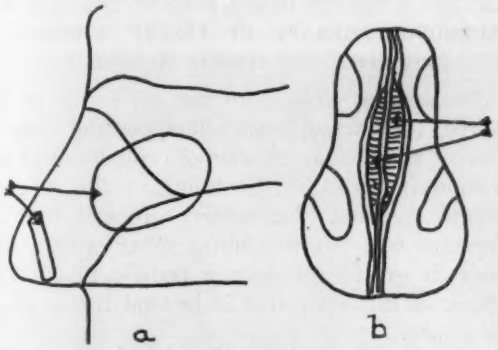

Fig. 2.—Implantation of cartilage strips. Arrows indicate the cartilage implants.

The preserved cartilage which was placed in sterile saline solution at the beginning of the operation is taken, and all sharp corners are rounded. The largest buckled piece of cartilage which will fit without too much tension on the mucoperichondrium is inserted between the mucoperichondrial flap and the septum in the area of the junction of the perpendicular plate of the ethmoid bone and the quadrangular cartilage. The cartilage is placed with the concave side against the septum and as high up toward the dorsum as possible. It is in this region that the greatest current of air passes during inspiration and expiration. If one has thoroughly separated the mucoperichondrial flaps, one or more large implants can be inserted on each side of the septum.

Two narrow straight oblong strips of cartilage are cut, the size varying according to each nose, and are implanted at the extreme anterior free end of the septum (fig. 2). The columella is resutured to the septum by two interrupted no. 1 black silk sutures. The sutures pass through the two anterior implants. The mucoperichondrium is tightly resutured to the vestibular skin by interrupted no. 000 catgut sutures. This step is important, and no drainage is permitted. No packing is necessary.

No undesirable complications or necrosis of the mucoperichondrial flaps has occurred after implantation of preserved cartilage. However, the cartilage was extruded in 3 instances: 1 in which the columella was not tightly sutured to the anterior free end of the septum, and 2 in which the mucoperichondrium had been buttonholed. If a mucoperichondrial flap is buttonholed, the operation on that side should be discontinued, the surgeon reoperating a few weeks later. However, the opposite side may be safely transplanted, without the extrusion of the implant. In 5 other cases there was no buttonholing and tight septocolumellar sutures were placed without extrusion of the implant.

After these cases had been observed for a period of eighteen months, it was found that some absorption of cartilage occurred in the septal implant. Moreover, the cartilage in this site appeared to be absorbed more rapidly than that transplanted into the dorsum for a saddle defect or into the columella to correct retraction. Marked improvement is obtained by this simple procedure, which takes about twenty minutes to perform. The odor disappears, but crusting may reoccur in a milder form. The nasopharyngeal scabbing diminishes, and the patient can more easily hawk up any postnasal accumulation. Better results are procured if rhinoplastic narrowing is performed at the same time.

Unilateral Moderate Atrophic Rhinitis.—The surgical procedure to be followed in cases of unilateral moderate atrophic rhinitis depends on both the history and the results of the clinical examination of the patient. If there is ample breathing space with no crust formation on the narrow side, this side should not be altered by any surgical procedure. The mucoperichondrium is elevated on the atrophic side only and preserved cartilage implanted as previously described.

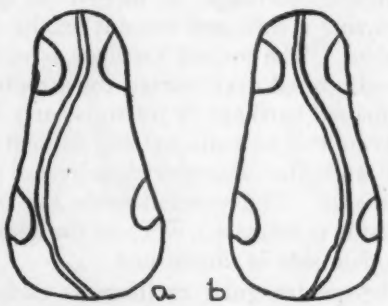

Fig. 3.—(*a*) Spoon-shaped septal deformity, with the convexity to the right. (*b*) Reversal of the septal cartilage, with the convexity to the left.

Should one side be extremely narrow, with insufficient breathing space, while the other is

wide, scabby and atrophic, either of two procedures may be used:

1. The classic approach for submucous resection is performed, the mucoperichondrium being elevated on both sides of the septum; or both mucoperichondrial flaps may be elevated by beginning at the anterior free end of the septum.

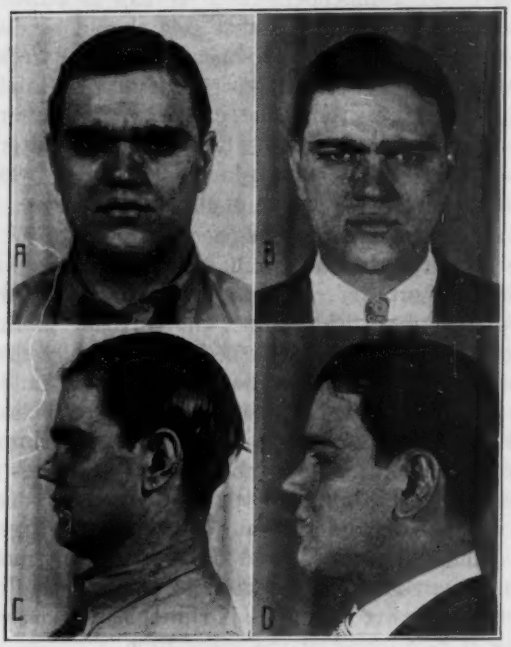

Fig. 4.—*A* and *C* are preoperative photographs. Note the typical broad saddle-shaped ozenic nose with retracted columella. *B* and *D* are postoperative photographs showing the result after two preserved septal cartilage transplants were placed in the dorsum, one in the columella, to depress it, and two in the septum.

By means of the swivel knife, as much of the quadrangular cartilage is incised as possible while leaving a sufficient amount on the dorsum for support. The incised cartilage is now fractured and pushed over toward the atrophic side, or the incised cartilage is fractured and wedged in between the remains of the dorsum of the septum and the mucoperichondrium on the atrophic side. The ventilation on the previous narrow side is increased, whereas the aeration on the atrophic side is diminished.

2. The quadrangular cartilage is excised and replaced in a reverse direction. This method was suggested by Dr. Alexander Laszlo, of the New York Post-Graduate Medical School and Hospital, and may be performed when the septum has a spoon-shaped deformity. The two mucoperichondrial flaps are elevated as is customary, and then, by means of the swivel knife, almost the entire quadrangular cartilage is excised in one piece, a sufficient amount being left for dorsal support. The cartilage is washed in sterile saline solution and is replaced in a reversed direction, the anterior free end being placed posteriorly. The convexity of the cartilage is now on the atrophic side; thus the latter side is narrowed and the ventilation of the stenotic side is increased. The mucoperichondrial flaps are resutured to the columella, and the nose is packed with sterile petrolatum-impregnated gauze. The packing is left in situ for forty-eight hours to keep the septum in place (fig. 3).

SURGICAL THERAPY OF SEVERE ATROPHIC RHINITIS OR TERMINAL OZENA

The surgical technic for the correction of the scarred, contracted, syphilitic-appearing nose is to make a sufficient number of cephalocaudal relaxation incisions in the lining of the nose to liberate the scar contracture. It will then be necessary to graft the lining either with a free Thiersch graft [10] or with a pedicle tube. The technic of this operation is beyond the scope of this paper.

SUPPLEMENTARY PLASTIC PROCEDURES TO DIMINISH THE INTAKE OF AIR

Additional procedures which may be used to diminish the intake of air are well known to the

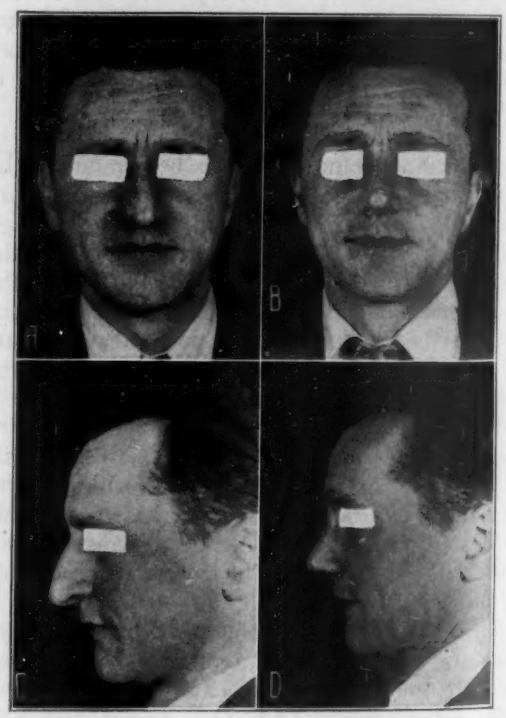

Fig. 5.—*A* and *C* are preoperative photographs. Note the nasal deformity associated with atrophic rhinitis. *B* and *D* are postoperative photographs showing the result of routine rhinoplasty: excision of the hump and narrowing of the lateral walls of the lobule and of the alar openings as in figures 7 and 8.

10. Gillies, H. D.: Deformities of the Syphilitic Nose, Brit. M. J. **2**:977, 1923.

rhinoplastic surgeon, who performs them routinely in narrowing the nose. If, in addition to atrophic rhinitis, the patient has a nasal deformity, a routine nasal plastic treatment tends to improve the condition. It may be noted that the majority of ozenic patients have a broad, slightly saddle-shaped nose with an upturned tip. For this type of patient, in addition to implanting a strip of preserved septal cartilage in the septum, it may also be desirable to implant a strip in the dorsum of the nose to correct the saddle deformity (figs. 4, 5 and 6).

Fig. 6.—*A* and *B*, post-traumatic saddle nose, a broad nose with wide nasal chambers and associated moderate atrophic rhinitis. *B* and *D*, postoperative rhinoplastic result.

Kopp [11] treats atrophic rhinitis by employing the classic procedure of narrowing the nose by sawing off the frontal processes of the maxilla and fracturing these bones medially by digital pressure. Wolf [12] claimed "gratifying results" following rhinoplasty in atrophic rhinitis. Joseph [13] performed rhinoplasty for atrophic rhinitis during the first World War with good results.

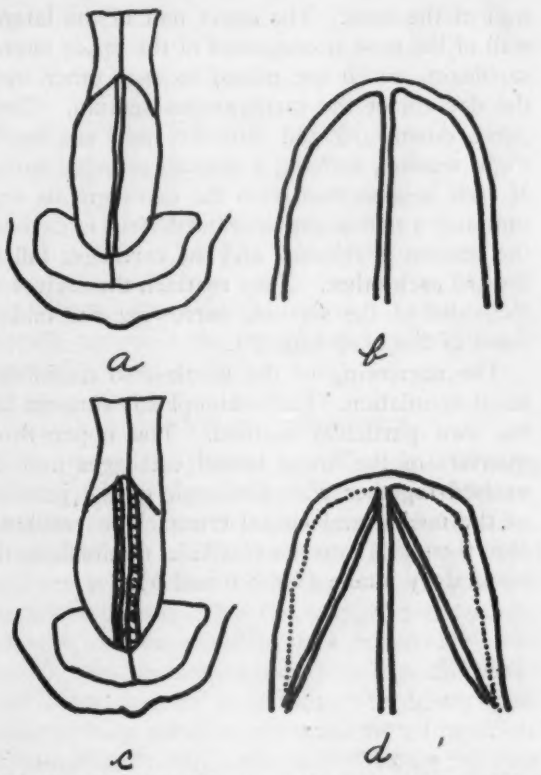

Fig. 7.—(*a* and *b*) Diagrammatic representations of the upper lateral cartilages. (*c*) Excision of a cephalocaudal strip. (*d*) Narrowed middle one third of the nose.

A study of the framework of the nose reveals how much the nasal chambers may be narrowed by rhinoplasty. The frontal processes of the maxilla are fractured medially after lateral osteotomy, narrowing the upper half of the lateral

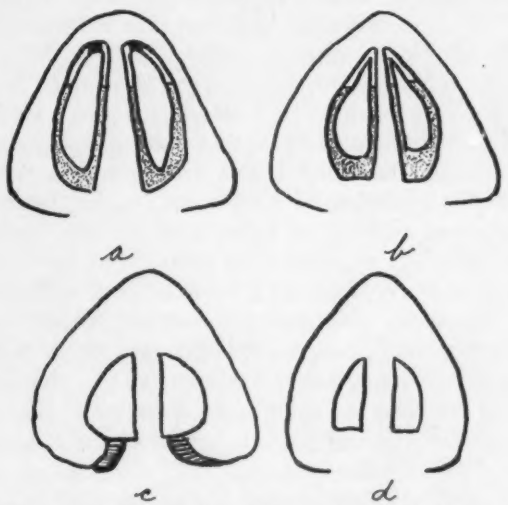

Fig. 8.—(*a*) Area of cartilage to be excised at the angle. (*b*) Narrowed lobule of the nose. (*c*) Excision of the base of the ala. (*d*) Narrowed alar openings.

11. Kopp, M. D.: Atrophic Rhinitis in Plastic Surgery, Laryngoscope **50**:510, 1940.

12. Wolf, G. D.: Rhinoplasty and Its Relation to Rhinology, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1941, p. 81.

13. Joseph, cited by Aufricht: Personal communication to the author.

wall of the nose. The lower half of the lateral wall of the nose is composed of the upper lateral cartilages, which are united to each other over the dorsum of the cartilaginous septum. They curve downward and laterally, and are under slight tension, forming a smooth rounded curve. If each is separated from the cartilaginous septum and a narow cephalocaudal strip is excised, the tension is released and the cartilages fall in toward each other. They reattach themselves to the sides of the septum, narrowing the middle third of the nose (fig. 7).

The narrowing of the lobule also diminishes nasal ventilation. Each rhinoplastic surgeon has his own particular method. The upper three quarters of the lower lateral cartilages may be excised together with the angle of the junction of the medial and lateral crura. The vestibular skin then sags into the vestibule, diminishing the inspiratory intake (fig. 8 *a* and *b*).

In those cases in which the alae are abnormally wide at the base, Joseph's method of excising a full thickness quadrilateral portion is ideal (fig. 8 *c* and *d*).

CONCLUSIONS

1. Preserved septal cartilage may be implanted on each side of the cartilaginous and bony nasal septum without necrosis of the mucoperichondrial flaps. Patients with moderate atrophic rhinitis and those with ozena obtain immediate relief by this procedure.

2. The results are better if, in addition to transplanting preserved septal cartilage into the septum, a rhinoplasty is performed.

3. The associated atrophy of the upper respiratory tract is also improved by the advocated nasal operation.

31 Lincoln Park.

MECHANISMS OF OCULAR MOVEMENT IN MAN

INFLUENCE OF THE VESTIBULAR APPARATUS

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CHICAGO

The otologist's ability to interpret vestibular movements of the eyes is directly related to his understanding of the various neural mechanisms responsible for ocular movements. As Gordon Holmes [1] has pointed out, in man vision is exclusively a cortical phenomenon, and the cortex in man is normally the seat of all reactions for ocular movements excited by retinal stimuli. Therefore it is necessary to study clinical material in attempting to understand the physiology of such movements in man. Since most of the otologist's observations are made on conscious subjects with cortical and visual function, he must be able to assess the role of the cortical and visual mechanisms controlling ocular movements when evaluating the role of the vestibular mechanisms in these movements. A description of some of the mechanisms illustrated by records of ocular movements [2] should help to orient vestibular mechanisms for these movements among the other mechanisms.

The objective of every movement of the eyes is clear macular vision. This is attained by keeping the foveas opposite the area of interest for a minimal time, about one-fifth second. The most restful or most easily maintained position for the eyes is central. In going from one point of fixation to another the eyes always move rapidly. Movement of the eyes may be (1) voluntary (frontal cortex), (2) reflex optic (occipital cortex activated by a retinal image) or (3) reflex vestibular (activated by end organs in the labyrinth). To meet all the demands for movement of the eyes, all three mechanisms must be working normally. Some movements result from a temporary preponderance of the activity of but a single center. Other movements represent the associated hyperactivity of several centers.

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From the Department of Surgery (Division of Otolaryngology) (Dr. Perlman) and the Department of Medicine (Division of Psychiatry), (Dr. Case), University of Chicago.

1. Holmes, G.: The Cerebral Integration of the Ocular Movements, Brit. M. J. **2**:107-112, 1938.

2. Perlman, H. B., and Case, T. J.: Nystagmus: Some Observations Based on an Electrical Method for Recording Eye Movements, Laryngoscope **49**:217-228, 1939.

VOLUNTARY MOVEMENTS

In the motor area of each frontal lobe there is a center for movement of the eyes which enables one to move the eyes away from the center voluntarily. This movement is always rapid and can be described as due to a stream of neural impulses flowing from the cortex to the ocular muscles concerned with this movement. The ocular muscles are thus voluntarily contracted and overcome the tonic muscle reflexes operating to keep the eyes in the resting midposition. These reflexes will alone slowly bring the eyes back to center. This observation can be made on an anesthetized subject. Good cortical function permits one to maintain the eyes firmly in a deviated position for some time. This is aided by the cortical fixation centers operating to maintain clear retinal images as from a point of fixation but can be carried out with no retinal images, as when the eyes are closed. A lesion of the frontal center on one side temporarily prevents voluntary displacement of the eyes to the opposite side of center but does not prevent reflex optic or reflex vestibular stimuli from moving the eyes into this same area.

The cortical centers not only enable the subject to move his eyes away from center and keep them there but enable him to make this movement rapidly from one position to another. This can be done under closed lids, that is, without retinal images, or it may be aided by vision—when fixation on a point in one area is rapidly followed by fixation on a point in the new area after the eyes have been moved. With or without retinal images, this voluntary movement is carried out very rapidly (in about one-twentieth second). The purpose of this rapid movement of the eyes from one point to another is to avoid blurred retinal images during the period of movement. The movement of the eyes is so rapid that no retinal images result. One can therefore see a clear image with the eyes in one position, and then another clear image after

moving the eyes rapidly to a new position. All day long the eyes move in this manner. If the point of interest remains too far from center, the head is turned rather than the eyes. The ability to move the eyes rapidly and accurately from one point of fixation to another is helped by good vision with associated adequate development of the cortical fixation centers. When vision is poor, this movement cannot be carried out as rapidly or as distinctly, because the eyes approach the point of fixation uncertainly and do not stop abruptly after reaching the new point of fixation but may swing beyond and around it. Cortical anoxia also impairs this ability of moving the eyes rapidly and accurately from one point of fixation to another.[3] The ability to maintain the eyes deviated away from center after this rapid movement is dependent on good vision and normal cortical and cerebellar stimuli. It is dependent on good vision in so far as this develops the occipital cortical centers which enable the subject to keep his eyes on a point of fixation. This is particularly concerned with the greater visual acuity at the macula as compared with the rest of the retina. The globe is fixed so that the object of interest is opposite the macula. This fixing power from the occipital cortex assists the frontal cortex in voluntarily maintaining the eyes in a deviated position, and after it has been well developed, it can normally operate even with no retinal stimulus, as when the eyes are closed. A subject whose fixation center is poorly developed because of defective vision will have difficulty in voluntarily maintaining the eyes in a deviated position. One may describe this in another way by saying that the subject cannot deliver a sufficiently strong stream of neural impulses from his cortex to the particular muscles of the eyes to overcome for long the tonic muscle reflexes which operate to bring the eyes slowly back to center. This cortical source of impulses can be said to become inadequate soon after its activation, enabling the eyes to be pulled slowly back toward center. The slow return movement results in blurred retinal images that momentarily reactivate the fixation centers in the occipital lobe which help the frontal cortex to move the eyes rapidly back to the point of fixation. A succession of such periods of adequate and inadequate cortical stimulation produces a rhythmic movement of the eyes, or nystagmus. The quick component results from a combination of stimuli from the frontal and the occipital cortex; the slow component is the lower tonic muscle reflex tending to bring the eyes back toward center when deviation occurs. Such nystagmus has the quick component in the direction of deviation. Cortical stimuli may become inadequate as a result of exhaustion, toxemia, the action of sedatives and other influences and produce this type of nystagmus. When such nystagmus is present because of poor vision, it can be altered or eliminated by entirely removing the retinal stimuli—as by closing the eyes. Now the periods between cortical recoveries are longer, and fewer attempts at lateral deviation are seen per unit of time, or no attempts may be made. Thus, even with poor vision the indistinct retinal images help the subject to maintain the eyes in a deviated position. The corticoretinal component of any nystagmus can be eliminated by closing the eyes. If nystagmus persists (as observed in the record), it indicates the activity of some lower mechanism or the persistence of a habit pattern. If the subject has been born with poor vision so that adequate power of the fixation center does not develop, nystagmus in the direction of gaze is most marked. In miners the failure to develop good fixation habits consequent on poor illumination results in similar nystagmus. Such nystagmus can be experimentally produced in a dog by keeping him in a dark room from birth.[4]

Once the cortical centers for fixation have been well developed, poor vision does not affect so much this power to maintain the eyes away from center voluntarily. Nevertheless many peripheral occular lesions can result in poor vision, poor functioning of fixation and fusion centers and difficulty in maintaining lateral deviation. Some persons with good vision and with no signs of neurologic disease can voluntarily move the eyes away from center but are unable to maintain the eyes in the deviated position or they display this inability only when the eyes are deviated in one direction. These persons have nystagmus in the direction of deviation and may be considered to have a congenital defect, such as an inadequate cortical center for maintaining the eyes in a deviated position, but are able to make frequent successive attempts to do this. These persons usually show no ocular movement in the central position.

The cerebellum is also concerned with this cortical ability to maintain the eyes in a position lateral from center. With cerebellar disease this power may be permanently lost. Sometimes it is more affected for movement toward the side of the lesion than for movement away,

3. McFarland, R. A.; Kuehr, C. A., and Berens, C.: The Effect of Anoxemia on Ocular Movements While Reading, Am. J. Ophth. **20**:1204-1221, 1937.

4. Lorento de Nó, R.: Personal communication to the author.

and is then reflected in the longer periods between the quick return movements to the desired deviated position to the side of the lesion. The cerebellum may be considered as the site of stimuli for muscle tonus and reciprocal innervation brought into action on changing the state of the ocular muscles. Normal symmetric function from both cerebellar lobes may be considered as essential for the correct tonic eye muscle reflexes to permit a maintained voluntary or cortical deviation of the eyes lateral from center. In cerebellar disease the reflex neural mechanisms (tonic eye muscle reflexes) for maintaining deviation are disorganized. The eyes tend to return to the central position. Cortical impulses are adequate to produce deviation but not to maintain it. The result is a succession of quick movements in the direction of gaze interspersed with slow pulls to the central position. As cortical impulses accumulate they become adequate to produce the characteristic quick movement. Rhythmic ocular movements, called nystagmus, are therefore seen. Lesions of the quadrigeminal plate interfere with vertical deviation of the eyes, resulting for example in inability to raise the eyes above center or inability to maintain them above center and a succession of slow movements back toward center and rapid ones up to the desired deviated position. Normal symmetric labyrinthine stimuli may also be necessary to maintain the eyes in a deviated position, but they are probably not definitely important in this respect, since almost all persons who have a chronic loss of function in one or both vestibular end organs can maintain the eyes apparently normally in lateral deviation, especially when the eyes are open and clear retinal images for the fixation reflex aid the cortex and the cerebellum. There normally is little demand to keep the eyes deviated away from center. It is much easier normally to move the head toward the new position of visual interest so that the eyes will remain near center. When some involuntary or reflex stimulus moves the open eyes away from center, there develops a cortical habit pattern of bringing them back rapidly toward a point of interest near center.

REFLEX OPTIC MOVEMENTS

Motor fibers that enable the eyes to turn toward an object in the periphery of the visual field, that control fusion, convergence and other activities, originate in the occipital or visual cortex. This center is also the source of motor fibers for the ocular muscles that are concerned with the eyes' ability to follow a moving object.[5] This is not a conscious rapid voluntary movement like that initiated from the frontal lobe, but a reflex movement, the speed of which is largely controlled by the speed of the moving object and requires retinal stimuli to sustain it. The retinal images need not be clear. This neural mechanism that makes the eyes follow a moving object can still operate even when the occipital cortex is destroyed, presumably through reflex activity at a subcortical level—initiated by moving retinal images.

The ability of the eyes to adhere to a point of fixation enables a person to see an object clearly when it or the subject is in motion. The subject fixes his eyes on a portion of the visual field in front of center and the eyes keep moving at the same rate as this area moves so that its light rays remain long enough on the retinas to produce retinal images. Then the eyes move rapidly to a new visual area apposite the central position of the eyes and the slow following movement of the eyes starts over again.

The rapid movement from one field to the new one is so brief that no retinal images result; thus the subject avoids the unpleasant sensation of blurred moving retinal images and presents to his visual cortex a succession of clear retinal images that assist him to orient motion. The movement of the eyes is called optic kinetic nystagmus. It may be disorganized by lesions of the occipital cortex.[6] The fixation reflex must be under cortical control to overcome it when necessary and permit the rapid return of the eyes toward center. For instance with certain cortical lesions the cortical control over the fixation reflexes is interrupted, and the eyes remain fixed on an object until the retinal images are interrupted. As the frontal cortical lesion impairs voluntary movement, the fixation reflex becomes accentuated. The patient cannot pull his eyes away from the area of interest. Movement to a new area of interest is accomplished by briefly closing the eyes. This breaks the fixation reflex by interrupting the retinal stimuli. This optic kinetic ocular movement cannot be carried out accurately if there is asymmetry in the neural stimuli from the vestibular systems. The resulting inaccuracy results in blurred retinal images, which give rise to a complaint of dizziness by the patient.

An example of normal cortical function moving the eyes through a small area rapidly and

5. Spiegel, E. A.: Physiopathology of the Voluntary and Reflex Innervation of Ocular Movements, Arch. Ophth. **8**:738-753 (Nov.) 1932.

6. Fox, J. C., and Couch, F. H.: Psychol. Monog. **47**:250, 1936.

accurately from one point of fixation to another is seen in reading. Here the head may remain motionless, the eyes being moved rapidly to four or six successive points of fixation to obtain retinal images for all the words on one line; then the eyes are moved suddenly and accurately all the way back to the beginning of the next line.

Ocular movements may be initiated directly from the frontal cortex or reflexly from the occipital cortex by retinal stimuli. The cerebellum cannot initiate ocular movements but is concerned with the tonus of the extraocular muscles.

REFLEX VESTIBULAR MOVEMENTS

The vestibular apparatus is not important for normal movement of the eyes in man. Most normal demands for movement of the eyes can be met by a subject with no vestibular function. Stimulation of the vestibular end organs can, however, initiate movement of the eyes. The fundamental ocular movement resulting from stimulation of the vestibular end organ is slow conjugate deviation away from center; it depends on bending of the hair cells by motion of the cupular substance in which they are embedded. The normal physiologic stimuli for this end organ are movements of acceleration and deceleration. It is a reflex phenomenon that has a short latent period. The ability to see clearly while moving the head (to move the eye accurately to keep it on a point of fixation while the head moves) is perhaps aided by the induced vestibular ocular reflex arising from accurately balanced vestibular mechanisms. In man most of this ability, however, is dependent on the optokinetic reflexes [7] and a little on the neck reflexes. With the eyes open, counter rolling of the eyes on moving the head occurs in the subject with no labyrinthine function as well as in the normal subject. With the eyes closed this counter rolling is disorganized in both the normal subject and the subject with dead labyrinths. When there is great asymmetry of the stimuli from the vestibular systems, fixation cannot be accurately maintained. This leads to blurring of the retinal images and contributes to the sensation of dizziness.

When there is sudden asymmetry in the stimuli from the two vestibular systems, the eyes are forced away from the resting position. Stimuli from the frontal cortex and from the fixation centers activated by retinal images may be strong enough to overcome this force working to move the eye. If, however, the cortical stimuli are unable to overcome the vestibular stimuli, fixation is not possible and blurred retinal images result.

As the eyes are deviated from the point the subject is attempting to fix, voluntary or fixing stimuli will be set up in the cortex to bring them back rapidly toward the point of fixation. This occurs regardless of the quadrant in which the eyes have been placed for this point. Thus the vestibular stimuli cause the eyes to move in a constant direction from the point of fixation. The cortical stimuli rapidly become inadequate, so that the stronger vestibular stimuli begin almost at once to pull the eyes away again. As the vestibular stimuli increase, fixation in any part of the visual field becomes impossible. The resultant subjective sensation is one of rotation.

When slow conjugate deviation from the vestibulo-ocular reflex occurs while the centers for cortico-ocular movement are inoperative and the eyes are open—as in light anesthesia or in disease of the cortical centers for ocular movement or of their tracts—no quick return movement of the eyes is observed, and lateral deviation may be maintained as long as the vestibular stimulus is operating. Indeed, the accentuation of the fixation reflex following frontal cortical lesions may keep the open eyes firmly opposite the point of fixation even when the vestibular impulse is strong. Inactivation of the fixation mechanism as by strong lenses will now permit vestibular stimuli to move the eyes slowly into the deviated position. The deviated position is maintained until the eyes are uncovered; then they return to stay in the central position. If the strong lenses are again placed over the eyes, the eyes will move slowly to the lateral position while the vestibular stimuli are active.

Pressure on a labyrinthine fistula in a patient under anesthesia will produce slow conjugate deviation of the eyes, positive pressure producing movement in one direction and negative pressure movement in the opposite direction. Momentary pressure with a probe on a labyrinthine fistula in a conscious subject will produce a brief conjugate deviation of the open eyes followed by a rapid return to the previous position—whether central or away from center. The latter movement is the result of stimuli from the fixation centers. Sustained pressure on the fistula with the eyes open in a conscious subject results in a series of slow deviations in one direction interspersed with a series of quick return movements in the opposite direction. One may describe the effect by considering that the vestibular stimulus sets up a strong vestibulo-ocular reflex forcing the eyes slowly away from center.

7. Fenn, W. O., and Hursh, J. B.: Movements of the Eyes When the Lids Are Closed, Am. J. Physiol. **118**:8-14, 1937.

The visual stimuli resulting from this slow movement while the head is motionless are blurred retinal images. These blurred retinal images set up central cortical reflexes in a conscious subject that cause a rapid return of the eyes to the point of fixation. This later stimulus becomes inadequate almost at once, and the stronger vestibular stimulus immediately begins again to move the eyes away slowly from this point.

When constant pressure is maintained on a fistula in a conscious subject whose eyes are closed, quick return movements may still occur. Often, however, the record does not show any movement. This observation suggests that a retinal stimulus may be important in activating the vestibular ocular reflex for initiating a primary movement. Artificial reduction of visual acuity as by strong lenses inhibits the fixation reflex, thereby allowing the vestibular movement of the eyes to operate for a longer time and with greater velocity. Quick return movements still occur. Wider and slower nystagmus is the result. The duration of the nystagmus is longer because the vestibular stimulus is not as readily overcome.

In the presence of spontaneous vestibular nystagmus, more marked movements of the eyes are observed when the subject deviates his eyes toward the side of the quick component than when he turns them in the direction of the slow component. This may be explained on the basis of superimposition of vestibular impulses (slow pull) on the tonic impulses tending to move the eye toward center. This results in the cortical stimuli, operating to maintain deviation, becoming more rapidly inadequate and leads to frequent and more quick movements. When such a person turns his eyes in the direction of the slow component, the eyes can be better maintained in the deviated position since the vestibular stimulus operating to move the eyes slowly in that direction is now working against the tonic muscle stimuli operating to move the eyes slowly back toward center. Therefore the cortical centers operating to maintain the eyes in the deviated position function more adequately, and the globes are more quietly maintained in that deviated position. If the final common pathways (posterior longitudinal bundle and others) for all ocular movements are affected in the pons, none of the ocular movement mechanisms may work because of paralysis of the ocular muscles. This may be a unilateral or a bilateral involvement such as occurs with pressure on or disease of the pons. If unilateral, the unopposed pull of the occular muscles on the nonparalyzed side may keep the globe deviated away from center and away from the side of the lesion and prevent any kind of movement (voluntary, optokinetic, vestibular) of the globe beyond center toward the side of the lesion. A peripheral lesion of the nerve for the ocular muscle may lead to similar results.

950 East Fifty-Ninth Street (Dr. Perlman).

APPENDIX

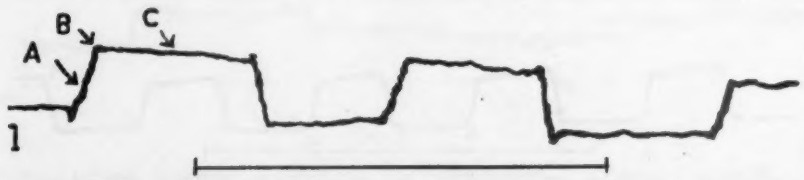


Fig. 1.—In this and all subsequent figures the bracketed straight line represents one second. Normal voluntary lateral ocular movements at a visual angle of about 12 degrees, looking rapidly between two objects. The subject makes about three sweeps of the eye in one second and keeps the eye well fixed on the object for about one-third second, or long enough to obtain a clear retinal image. He moves the eye to the object on the opposite side in about one twenty-fifth second (*A*) so rapidly that no retinal image results during the time he is bringing the eye from one object to the next. The amount of time he fixes on the object varies a little but the time that the eye faces the object is long enough to see it clearly (*C*) and is six to ten times as long as the time taken to bring the eye over from one side of center to the other.

This is an example of frontal cortical conscious movement of the eye. The eye can be moved rapidly to the desired point and stopped accurately when the point is reached (*B*). Then the eye is held firmly opposite the point until a clear retinal image is obtained (*C*). The ability to keep the eye voluntarily deviated away from center is an expression of cortical activity. The ability to stop the eye accurately and abruptly when the point is reached and to maintain the eye opposite this point is aided by good vision with good fixation habits. If vision is poor and fixation is imperfect, the eye will approach the point more slowly and will not stop abruptly at the point but swing around it. Then, because the eye does not see the point clearly and the power of fixation is poor, it will almost at once begin to swing slowly away from the point and toward a central position. The number of times the subject with poor vision can move his eyes back and forth in one second is also reduced.

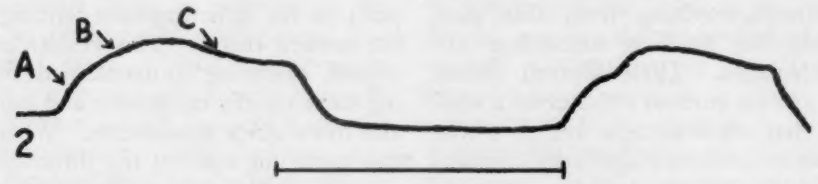


Fig. 2.—The slow, imperfect voluntary lateral movement of an eye in a child with poor vision and poor habits of fixation. The period for the rapid movement *A* is slower. The eye does not stop abruptly at *B* and is not firmly maintained at *C*.

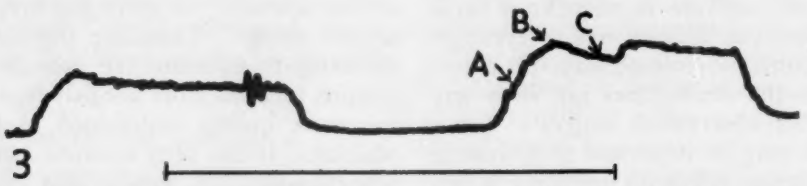
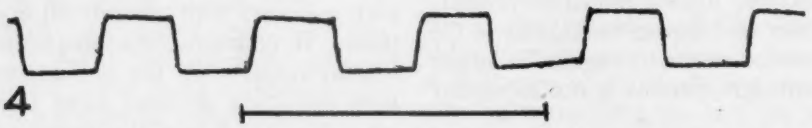


Fig. 3.—Movement of an eye of a normal subject with eyes closed. Even in the normal subject, when the eyes are closed the movement is slower and less accurate.

Cortical anoxia will also interfere with rapid accurate voluntary movements of the eyes or ocular movements that have been learned, as for reading.



Figs. 4 to 6.—The ability to move the eyes rapidly between two objects is not dependent on function of the labyrinths and can be accurately carried out even when there is spontaneous nystagmus.

Figure 4 records the accurate frontal cortical to and fro movement of an eye in patient L, between attacks of Ménière's disease.

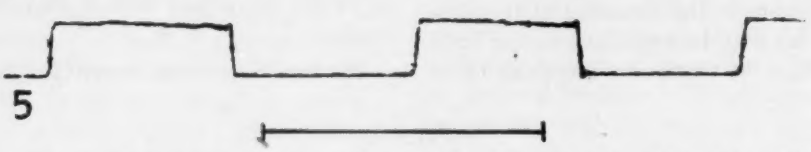


Figure 5 shows the accurate movements of an eye in a patient with good vision and good frontal cortex, observed in the face of spontaneous nystagmus.

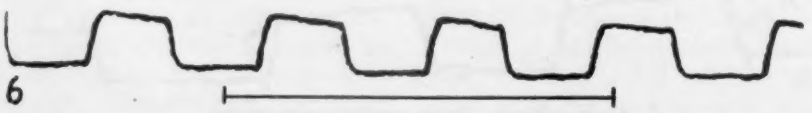
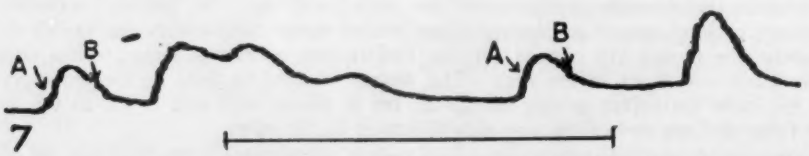
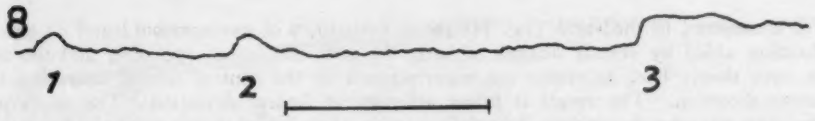


Figure 6 demonstrates accurate rapid to and fro movements of the voluntary frontal cortical type not affected by absence of function of the labyrinth on the right.

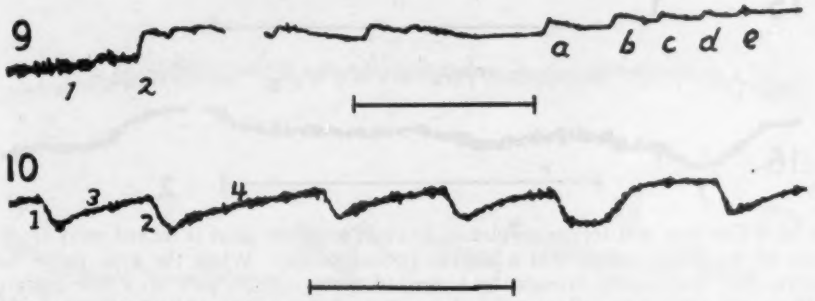


Figs. 7 to 10.—The ability to maintain voluntarily lateral deviation of the eyes for a long time (many seconds) is aided by good vision with good function of the center for fixation and normal symmetric cerebellar function. Normally the eyes can be kept in lateral deviation for many seconds without evidence of tiring.

A patient with congenital amblyopia was asked to move his eyes to the left and maintain them in that position. He made an attempt, producing a rapid lateral deviation (*A* in fig. 7). Poor vision and poor fixing power prevent him from keeping the eyes in the deviated position. The frontal and the occipital cortical stimuli become inadequate almost at once, and the eye returns toward center (*B*). Then the cortical centers recover, their stimuli again become adequate and again the eye is rapidly deviated to the left (*A*). A succession of ocular movements results (nystagmus in direction of gaze).

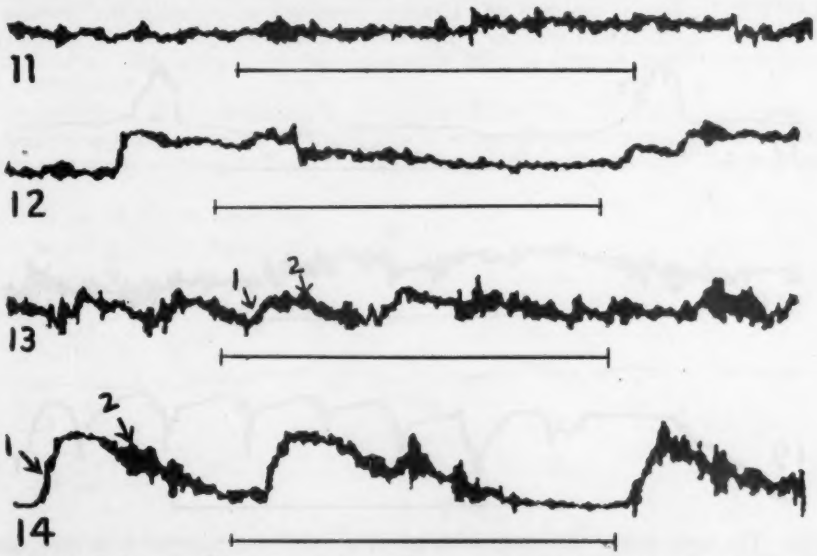


Sometimes the cortical centers recover less rapidly and there are longer intervals between attempts at lateral deviation. In figure 8 note the distance between *1* and *2*. (The patient has Schilder's disease. He was asked to move his eyes to the left and keep them deviated.) At *3* he has done better. Sometimes a subject may begin a well maintained lateral deviation, and after a few seconds the cortical stimulus becomes inadequate and permits the eye to return toward center. As this is taking place the cortex recovers and rapidly moves the eye back to the desired deviated position.



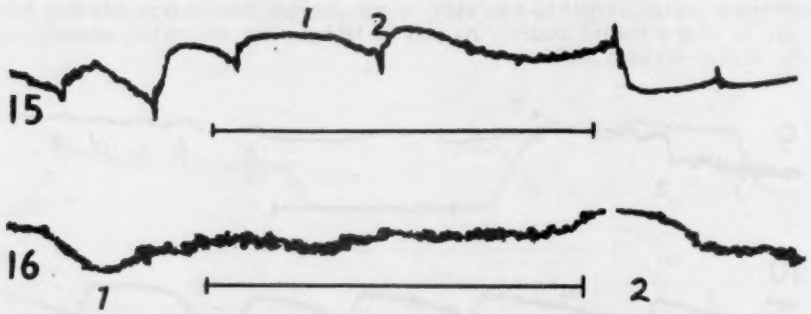
Cerebellar disease may impair the ability for maintaining the eyes in a deviated lateral position even in the presence of normal cortical function. There is no difficulty in keeping the eyes quiet in the midline.

At *1* in figure 9 the eyes of a patient with a cerebellar lesion are quiet in the midline. At *2* the subject voluntarily moves his eyes to the left, away from the side of his lesion, and although he has fairly good vision and no pathologic involvement of the frontal or of the occipital cortex, he cannot maintain the lateral deviated position. The eyes begin to move back to center, and as this occurs the cortical stimuli are renewed and move the eyes back rapidly to the desired deviated position. A spontaneous nystagmus in the direction of gaze results. He makes five attempts to deviate his eyes (five small excursions—*a, b, c, d, e*) in one second. Looking to the right (toward the side of his lesion), he brings the eyes over farther (*1* and *2* in fig. 10) but cannot maintain them there. They immediately start moving slowly toward center (*3* and *4*). The cortex requires more time to become adequate and makes fewer attempts to deviate the eyes (*2* per second).



Figs. 11 to 14.—Spontaneous nystagmus of the vestibular type may be arrested or reduced by the frontal cortical centers, assisted by activity of the fixation centers in the occipital cortex, even when the eyes are deviated away from center. Figure 11 illustrates how in a case of spontaneous nystagmus fixation, through good vision, keeps the eyes at rest in the midline, and figure 12, how it keeps them firmly in the deviated position. With the eyes in midline but closed, so that the centers of fixation that previously were activated by retinal images are no longer acting (fig. 13), the eyes begin to move in response to the lesion producing nystagmus (quick movement to the right, *1*, and slow movement to the opposite side, *2*).

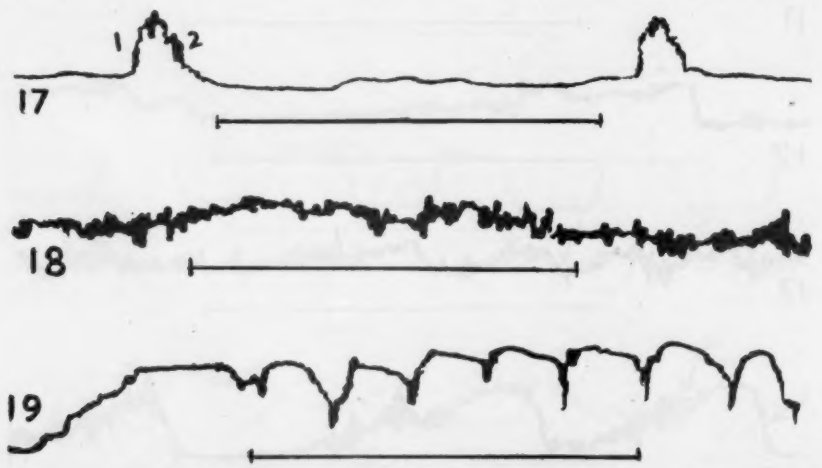
With the eyes closed and to the right (fig. 14), wide excursions of nystagmus (*1* and *2*) are recorded because the power of fixation aided by retinal images to keep the eyes still is not operating and the tonic reflexes acting to bring the eyes slowly back to center are superimposed on the central stimuli operating to move the eyes slowly in the same direction. The result is fewer attempts at lateral deviation. The appearance of this nystagmus when the eyes are closed suggests its origin outside of or below the corticoretinal system.



Figs. 15 and 16.—The eyes will follow a point of fixation when the head is moved away from it (*1* in fig. 15), to keep its image on the fovea and prevent a blurred retinal image. When the eyes move too far away from center for comfort, they are rapidly brought back toward center (*2*) to pick up a new point of fixation. This may be repeated several times when the head is being turned from one side to the other. A similar type of ocular movement occurs during linear movement either of the subject or of the environment. The normally innervated eye can maintain this type of movement accurately for hours without fatigue (optokinetic nystagmus) and may even continue to move in this manner for a short time after the stimulus has ceased.

Violent turning of the head initiates optokinetic nystagmus but probably also produces a vestibulo-ocular reflex as acceleration and deceleration of the head and of the fluid in the labyrinths take place.

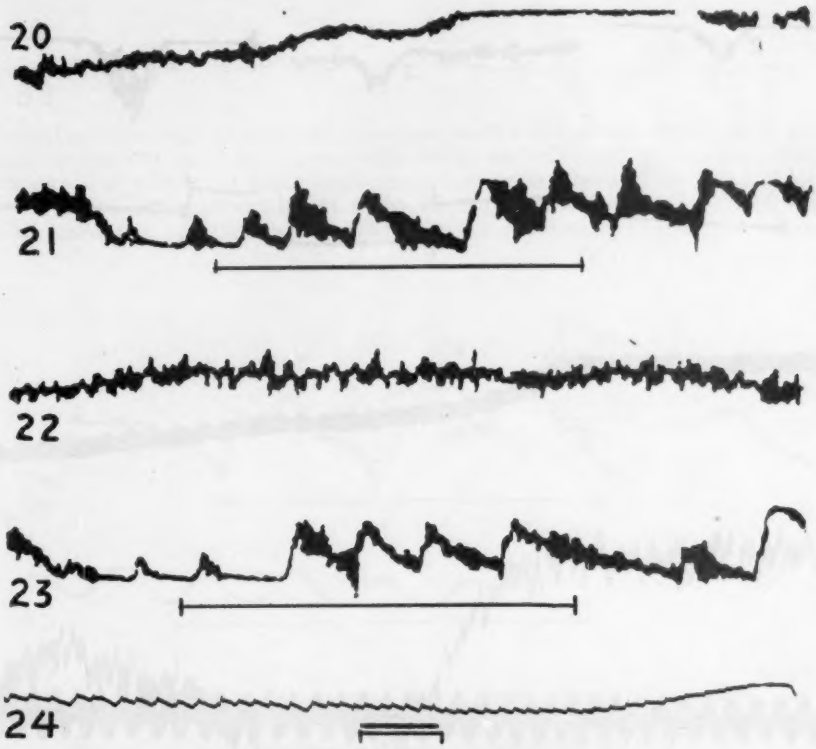
The vestibular component of the ocular movement during violent turning of the head can rarely be detected by recording with the eyes closed (fig. 16). The vestibular component is a small single slow deviation for each wide turn (*1* and *2*) of the head. It probably assists the open eyes to move accurately and to remain on the point of fixation so as to prevent blurred retinal images, although practically all of this ability is dependent on the optokinetic reflex. Often the pattern of the ocular movement on turning the head widely with eyes closed is practically identical with the pattern of movement when the eyes are open and indicates the formation of a habit pattern of movement.



Figs. 17 to 26.—The basic ocular movement initiated by a vestibular stimulus is a slow conjugate deviation away from center. It can be elicited by brief pressure on a labyrinthine fistula, when the eyes can be made to move for a short time in this way. In an anesthetized person only wide slow deviation of the eyes occurs when pressure is applied to a labyrinthine fistula.

Figure 17 records the brief conjugate deviation (*1*) and rapid return to the previous position of fixation (*2*) caused by momentary pressure on a labyrinthine fistula in a conscious subject with eyes open.

This vestibulo-ocular stimulus may keep the eye in a deviated position when a retinal stimulus from the open eye is added and the cortical centers for ocular movement are not functioning (i. e., under anesthesia or in disease of the cortical centers for ocular movement or their tracts).

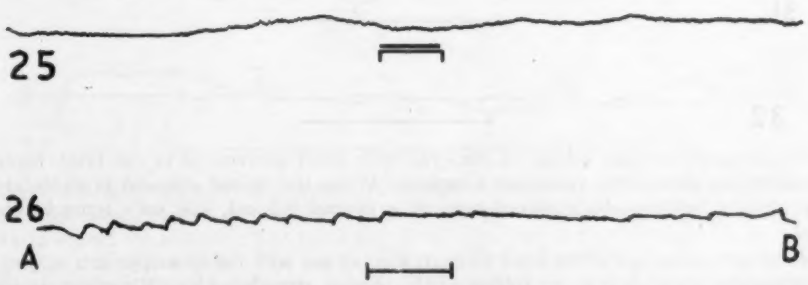


For figure 18 pressure is made on a labyrinthine fistula in patient L while his eyes are closed. No ocular movement is recorded. Figure 19 shows that when pressure is made on the same labyrinthine fistula while patient L is conscious, with eyes open, the pressure, aided by the retinal stimuli, produces a series of ocular movements. The slow deviation from the vestibular stimulus causes a blurred retinal image. This initiates another attempt at fixation (a cortical phenomenon) that brings the eye rapidly back toward center. The neural cortical stimulus for fixing the eye is not strong enough to overcome permanently the vestibular stimulus forcing the eye to the deviated position, and again a slow movement occurs to be followed once more by a quick return movement as the centers for this movement recover. A succession of slow lateral movements of the eye and quick return movements results. The direction of the movements remains constant regardless of whether the eye is at center or away from center.

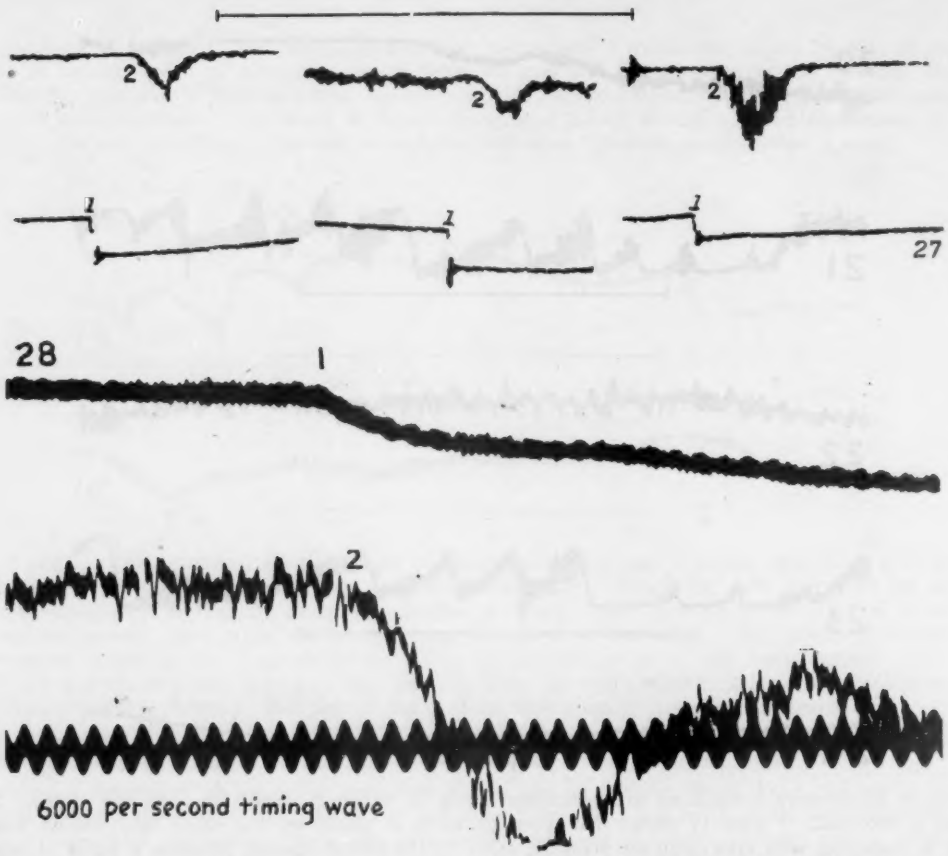
Maintained pressure on a fistula in patient C with eyes closed (fig. 20) causes no ocular movement. The same stimulus when the eyes are open (fig. 21) produces a series of lateral deviations and quick return movements. The latter movements are initiated by a blurred moving retinal image and are due to neural impulses from cortical centers.

Figures 22 and 25 are similar to figure 20.

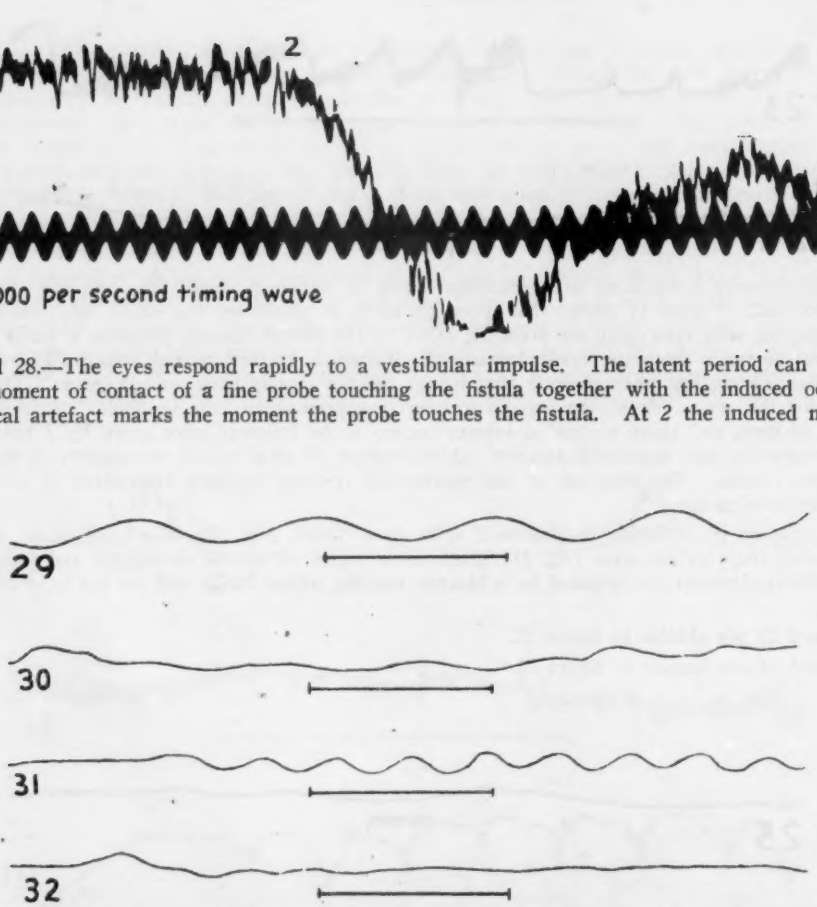
Figures 23 and 24 are similar to figure 21.



Nystagmus does not continue but gradually dies out after about ten seconds even though constant pressure is maintained on the fistula. Deviation of the cupular substance cannot be indefinitely maintained. A constant degree of slow losing of fluid past the cupular substance probably allows equilibrium to be established, and the cupular substance returns to midline. This is illustrated in figure 26. Constant pressure in the mastoid cavity began at *A* and stopped at *B*. Nystagmus, at first marked and rapid, finally disappeared after about ten seconds. (See also fig. 24.)



Figs. 27 and 28.—The eyes respond rapidly to a vestibular impulse. The latent period can be measured by recording the moment of contact of a fine probe touching the fistula together with the induced ocular movement. At *1* an electrical artefact marks the moment the probe touches the fistula. At *2* the induced movement of the eye begins.



Figs. 29 to 32.—Accurate counter rolling of the eyes with small movements of the head, mainly an optokinetic reflex, is not impaired by absence of vestibular function. When the retinal stimulus is obliterated by closing the eyes, this counter rolling becomes disorganized even in a normal subject, and only irregular movements of the eyes are recorded.

With slow side to side movement of the head through a small arc with the eyes open in a subject whose labyrinths are dead after meningitis, accurate counter rolling of the eye is recorded (fig. 29) which is identical with that seen in a subject with normal labyrinths. With the eyes closed (fig. 30), the counter rolling almost disappears. Only a few disorganized movements of the eye are recorded. The lack of a retinal image has obliterated or disorganized the optokinetic reflex. On more rapid movement of the head of the same subject with the eyes open (fig. 31), accurate counter rolling of the eye takes place. With the subject's eyes closed and retinal images lacking (fig. 32), this rapid movement of the head no longer leads to a synchronized ocular movement of the optokinetic reflex.

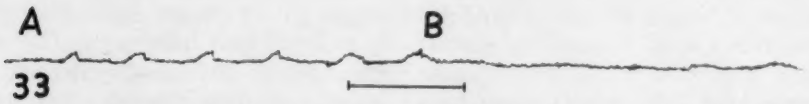
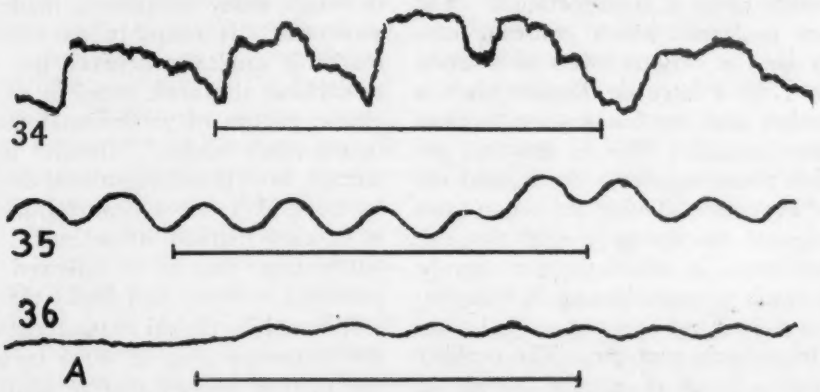


Fig. 33.—A retinal stimulus may be necessary to activate the vestibulo-ocular reflex. With the retinal stimulus removed by closing the eye, the vestibular stimulus may not be strong enough to initiate a primary movement of the eye. *A* is the record of a series of single displacements of an eye by a rapid series of discrete pulses of pressure on a fistula in the horizontal canal. At *B* the subject closes his eyes, and although the pulses of pressure continue at the same rate and the same intensity as when the eyes were open, no movement of the eye is recorded.



Figs. 34 to 36.—The ability to keep the eyes open and at rest even in the central position is dependent on the cerebral centers controlling fixation. When these centers receive inadequate oxygen or are poorly developed, as in congenital amblyopia, oscillatory movements of the eyes may occur even in the central position. These represent continued imperfect attempts at bringing the more sensitive areas of the retinas opposite the point to be visualized. These attempts at fixation cease when no stimuli are reaching the retinas, as when the eyes are closed. With no retinal stimuli these subjects may make little or no attempt to move the eyes away from center (as under the closed lids).

Failure of the cortical fixation and fusion centers due to lesions of muscles of the eyes or to refractive errors will also be reflected in an inability to keep the eyes open and at rest in the midline. Simultaneous stimuli from two eyes may be adequate to keep the eyes at rest, while with one eye closed the action of the fixation centers may be inadequate.

Figure 34 shows the midline oscillation of the right eye when the left eye is closed in a patient with congenital alternating strabismus who can keep the eyes at rest when both are open. When the eyes are closed, no retinal stimuli arise to produce an attempt at fixation, and the eyes remain quietly in the midposition.

Figure 35 records the bilateral midline oscillations of the eyes of a patient with congenital amblyopia when the eyes are open. These may be described as "searching movements." With the eyes closed (*A* in fig. 36), movement stops.

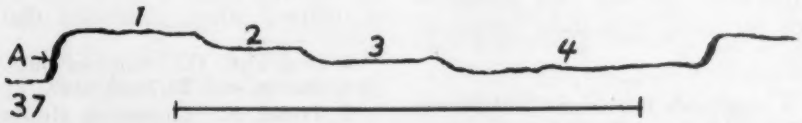


Fig. 37.—Complicated accurate cortical learned ocular movements produced for reading. At *A* the eye moves rapidly and accurately from the end of one line to the beginning of the next. Four periods of fixation (*1, 2, 3, 4*) are required to see all the words in that particular line. The eyes are moved rapidly, accurately and minutely to each successive area along the line. The rapid movement (one-twentieth second) produces no visual stimulus; the sustained position endures just long enough (one-fifth to one-half second) to produce a retinal stimulus and a cortical impression.

AVIATION DEAFNESS

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Aviation is the newest mode of transportation and during the past several years has rapidly gained a prominent place in passenger miles covered. There are medical problems in aviation as in every other form of transportation. The most frequent problems which confront otolaryngologists are the various forms of aviation deafness. In 1783 Pilatre de Rozier made a balloon ascension and on his descent noticed a rather severe earache. This is the first recorded instance of an injury to the human ear as a result of aviation.[1] Today the organ most frequently affected by flying is still the ear. There are two ways in which the ear may be affected as a result of participating in aviation: one is by the noise, and the second is by the changes in atmospheric pressure. The problem of aviation deafness was recognized during the last war, and a large amount of research was done.[2] It has been shown for many years that men who in their occupations are continually exposed to loud noises eventually become hard of hearing.[3] An example of this is the deafness that occurs in blacksmiths, recognized as a clinical entity in the otologic texts of 1890 to 1900 and called boilermaker's deafness. With the invention of the audiometer, considerable research was done showing the destructive effects on hearing of noise in various industries and occupations. With the advent of the low pressure chamber, investigations were made on changes in atmospheric pressure and in the oxygen content of the air and their effect on the human ear. Advances in aviation and especially in aerial warfare within recent years have intensified interest in the ear and have prompted further research by many prominent otolaryngologists.

Following World War I, Gradenigo[4] made a survey of Italian aviators who served in that war and described the hearing loss in the fourth and fifth octaves. Troina[5] in 1933, after a thorough study of airmen, made the following statement; "It seems to me there exists a remarkable similarity between the clinical picture of cochlear deafness, noted in aviators, and the classic picture of professional deafness peculiar to the noisy trades." Bunch[3] in 1937 demonstrated, first, that occupational deafness produced by noise is a nerve deafness and, second, that it is most marked immediately after excessive stimulation, that it is followed by a variable period of recovery and finally that an audiogram will reveal its typical characteristic sharp loss in the frequency area of 4096 cycles per second. He further proved that if stimulation is continued, the audiographic defect is apt to become steeper and broader, but with nearly normal hearing remaining for the lower tones. In 1938 Firestone[6] reported an audiometric study of hearing in a series of 109 pilots. He included only pilots who had from seven to twenty years of flying experience. His study demonstrated that the noise and the vibration incidental to flying are injurious to an aviator's inner acoustic apparatus. He expressed the belief that the audiographic studies, along with other functional tests, show symptoms pathognomonic of otosclerosis. The amount and the extent of the injury were found to be in direct proportion to the flying time. Firestone suggested the term "aero-otosclerosis" for the condition. Dickson, Ewing, and Littler,[7] after examining a number of military pilots, concluded that engine noise,

Presented as a candidate's thesis for the American Laryngological, Rhinological and Otological Society in June 1944.

1. Simpson, J. F.: A General Survey of Otorhinological Considerations in Service Aviation, J. Laryng. & Otol., **57**:1-7 (Jan.) 1942.

2. Bauer, L. H.: Medicine and Aviation, Tr. Am. Acad. Ophth. **46**:43 (Jan.-Feb.) 1942.

3. Bunch, C. C.: Symposium: Neural Mechanism of Hearing; Nerve Deafness of Known Pathology or Etiology, Laryngoscope **47**:615-691 (Sept.) 1937.

4. Gradenigo, G.: Funzione tubaria ed aviazione, Arch. ital. di otol. **30**:24-30, 1919.

5. Troina, F.: Changes in Hearing of Air Pilots: Statistical Study, Valsalva **9**:337-353 (May) 1933.

6. Firestone, C.: Bone Conduction in the Experienced Pilot and Probable Interpretation, Laryngoscope **48**:168-175 (March) 1938.

7. Dickson, E. D. D.; Ewing, A. W. G., and Littler, T. S.: Effects of Aeroplane Noise on Auditory Acuity of Aviators: Some Preliminary Remarks, J. Laryng. & Otol. **54**:531-548 (Sept.) 1939; in 1940 Year Book of Eye, Ear, Nose and Throat, Chicago, The Year Book Publishers, Inc., 1941, p. 339.

compared with other noise, produces a definite loss in the high tone range, usually in the region of 4096 cycles. They reported that the loss at first is only temporary but that it gradually becomes permanent, thus corroborating the observations of Campbell and Hargreaves.[8] The most effective protection, according to these investigators, is close fitting telephone receivers and an aviation helmet. Campbell and Hargreaves stressed the fact that there are great individual variations in the effect of flight on the hearing of various persons. This is due to the fact that in one person the mechanism is sensitive to fatigue as a result of insult, while in another a large amount of insult produced by noise will cause practically no hearing defect. They suggested that this may be the result of a familial tendency. In their experience a small permanent notch in the curve of hearing often appeared after about two hundred and seventy-five hours of open cockpit flying in planes having a powerful motor. They suggested further that the fatigue factor might be exaggerated by anoxia of the structures of the end organ of hearing.[8]

Following World War I, in 1919, Scott[9] reported a form of aviation deafness resulting from changes in pressure in the middle ear which produced an "invagination" of the tympanic membrane. In 1937 Armstrong and Heim[10] made a comprehensive study of the effect of changes in barometric pressure on the middle ear. In 1940 Campbell and Hargreaves[8] reported experiments showing that rapid descent without ventilation of the eustachian tubes results in a loss of hearing for the lower tones in the conversational range.

Today the problem of aviation deafness is fairly well understood as to causes, prognosis and treatment. The great increase in speed and maneuverability of modern airplanes, the greater altitude of operation, the marked increase in length of flight and the greater horsepower of the motors represent the main advances in modern aviation. With these advances the problems of physicians have been greatly increased. More than 80 per cent of the complaints associated with routine military aircraft flight are otorhinologic in origin. Furthermore, in about 70 per cent of these cases of discomfort the trouble has its origin in the ear. A recent survey by Tuttle[11] shows that with one of our established airlines, carrying a total of 1,840,190 passengers over a three year period, there were 7,111 cases of discomfort from all causes, and only 826 cases of trouble in the ears. That means an incidence of 0.8 per thousand. This report, of course, refers to routine commercial aircraft carrier service and is to be differentiated from military aviation. Such cases are not encountered frequently in commercial aviation but are much more frequently seen in military aviation. This is due to the facts that military aircraft are not soundproofed as are commercial planes, higher horsepower is used, greater speeds are attained, the rates of ascent and descent are much greater, and operations at greater altitudes are common. Tuttle and associates studied 200 civil airline pilots and found that in comparing their data with those obtained by Bunch on large groups of nonflying subjects, comparable in age, the pilots showed a loss from 10 to 15 decibels greater. Greene[12] reported approximately the same figures for another group of airline pilots. The evidence is inconclusive that any advanced degree of occupational deafness occurs in pilots. This appears to be true even in the older ones who have been exposed to the noise of motors for many hours of flying in planes with open cockpits.[13]

CLASSIFICATION

Aviation deafness for all practical purposes can be classified as aviation perception deafness or nerve deafness and aviation pressure deafness or conduction deafness. In the first type there are fatigue and injury of the inner organ of hearing. In the second type there results a defect in the ability of the structures of the middle ear to properly transmit sound from the external canal to the inner acoustic mechanism. Either of these types may be acute or chronic. At the School of Aviation Medicine at Randolph Field, Texas, Campbell and Hargreaves[8] undertook a thorough investigation of the subject from various angles and classified deafness of aviators as follows:

1. Acute fatigue of the end organ of hearing and related structures.

8. Campbell, P. A., and Hargreaves, J.: Aviation Deafness—Acute and Chronic, Arch. Otolaryng. **32:** 417-428 (Sept.) 1940.

9. Scott, S.: The Ear in Relation to Certain Disabilities in Flying, Reports of the Air Medical Investigation Committee, Medical Research Committee, Special Report Series, no. 37, London, His Majesty's Stationery Office, 1919, pp. 29-51.

10. Armstrong, H. G., and Heim, J. W.: The Effect of Flight on the Middle Ear, J. A. M. A. **109:**417-421 (Aug. 7) 1937.

11. Tuttle, A. D.: Safety and Comfort Aloft, J. Aviation Med. **10:**1-15 (June) 1939; The Role of Aviation Medicine in the Development of Aviation, Mil. Surgeon **85:**285-301 (Oct.) 1939.

12. Greene, R.: An Aviator and His Ears, Indust. Med. **7:**669-671 (Nov.) 1938.

13. Poppen, J. R.: The Ear in Flying, Laryngoscope **51:**974-982 (Oct.) 1941.

2. Chronic accumulative fatigue of the end organ and related structures.
3. Conduction deafness, due to changes in pressure in the middle ear.
4. Chronic conduction deafness, due to alterations of tissue resulting from faulty ventilation of the middle ear.

If there were always proper ventilation of the middle ear, one's only concern with aviation deafness would relate to that due to the effect of noise. In this case the noise would produce fatigue, which in time, if the effect was severe, would result in chronic accumulative perception deafness. However, there are circumstances in which the middle ear does not properly ventilate during changes of atmospheric pressure, and it is in such cases that one encounters changes which produce a picture of conduction deafness. These changes are brought about by a differential of pressure in the middle ear and in the external atmosphere which limits the mobility of the tympanic membrane and therefore its sensitivity to sound vibrations. These two conditions may occur in the same person, and an audiometric study may show both and result in a faulty interpretation unless one has in mind the two conditions, which may result from altogether different causes.

AVIATION PERCEPTION DEAFNESS

For many years it has been known that many types of sounds are injurious to the human ear. Riveters, boilermakers and similar groups have been examined and it has been found that they suffer from such noises as occur in their occupations. Aviation noises are chiefly those from engine explosions, propeller hum, sounds produced by wind and slip-stream effects on the structure and sounds from moving parts.[14] There are, of course, preaudible and suprasonic vibrations, which have not been clearly investigated. Much time and money have been spent to overcome these sources of noise. The sound from a whirling propeller increases as the speed of the tip of the propeller approaches that of sound. Therefore, the only means of reducing this mode of sound production is to reduce propeller peed. Much has been done in this field by making propellers larger and changing their pitch and airfoil so as to make them more efficient at given speeds. Up to the present time no satisfactory method has been found to decrease the noise associated with the operation of the engine or multiple engines. Such noises come primarily from the exhaust stack, and unfortunately all mufflers designed to date have reduced the horsepower too much to be of practical value.

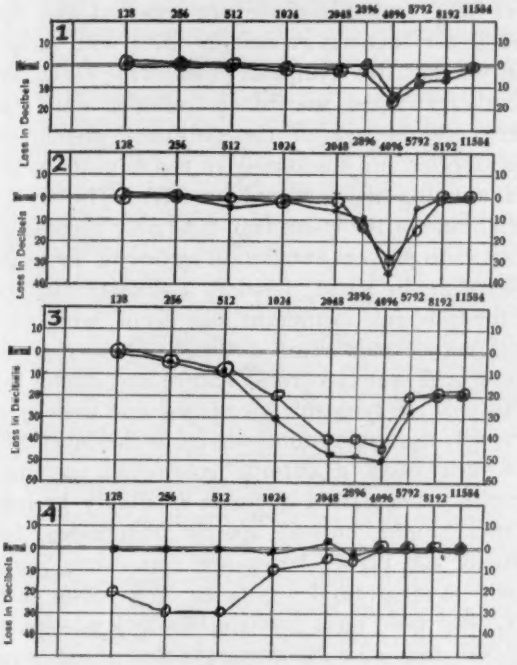
It has been shown that fatigue of the end organ of hearing is produced by the various sounds just referred to and is responsible for deafness in aviation, producing a definite hearing loss in the area of 4096 cycles.[8] If the fatiguing force is continued over a longer period, it is found that there is a fanwise spreading of the involved area until the conversational areas are encroached on. Howell defined fatigue "as a more or less complete loss of irritability brought about by functional activities." Fatigue is in direct proportion to the strength and the length of exposure. Recovery from the effects of fatigue after the fatiguing force is removed is in proportion to the strength and the duration of the application of the fatiguing force. Thus, one can see that if a fatiguing force is applied for a short period, there will be an effect produced from which recovery can be expected in a short period. If, on the other hand, the fatiguing force has been of great intensity or of long duration, one may expect a longer period of recovery. This results in what might be termed chronic accumulative fatigue and fits into the usual conception of traumatic perception deafness.

An analysis of airplane noise has shown that the frequencies of the highest intensity, 110 to 115 decibels, are at the low end of the acoustic spectrum.[14] This would suggest that a low tone was causing a high tone deafness. There is no agreement as to why this should occur. The explanation is not clear, but it is probable that a complex noise of high over-all intensity inflicts damage by excessive sound pressure on the most susceptible part of the cochlea, i. e., the basal coil. The basal coil is situated just opposite the round window, and this may explain why there is a loss in the 4096 cycle area, which is supposed to be located in the basal coil. Another explanation for this dip in the 4096 cycle area is that it may be due to the fact that the external auditory canal has in its normal condition a characteristic natural period which may act as a resonator intensifying a frequency in a complex sound in the region of 3000 cycles and thus cause a dip at 4096 cycles.

My duties as a licensed pilot and also medical examiner for the Civil Aeronautics Authority for the past seven years have given me an opportunity to observe many cases of the type described. I have flown on the airways, using aviation radio under severe static conditions, and have on a number of occasions run audiometric tests following such trips and noted the effects

14. Dickson, E. D. D.: Aviation Deafness and Its Prevention, J. Laryng. & Otol. **57**:8-10 (Jan.) 1942.

of noise. Audiogram 1 in the accompanying chart shows a defect found following a two hour flight in a 240 horsepower open cockpit airplane. Audiogram 2 shows the effect of engine and airplane noise following a four hour flight. It will be noted in each instance that there was a definite loss in the 4096 area, more marked in the longer flight. Fortunately, this loss was only temporary; hearing tests made the following day showed absolutely no defects. Audiogram 3 was made for a commercial pilot with slightly over eight thousand flying hours, most of them in open cockpit ships as an instructor. One notes here a marked dip in the 4096 range, which has spread and involved not only the 2048 area but



1, hearing defect following a two hour flight in a 240 horsepower open cockpit airplane. Pilot W. K. E., aged 22.

2, hearing defect following a four hour flight in a 240 horsepower open cockpit airplane. Pilot W. K. E., aged 22.

3, hearing defect in a pilot with eight thousand flying hours, most of which has been in open cockpit ships. Pilot A. W. W., aged 45.

4, hearing defect of a bombardier student sixteen hours after a practice bombing mission at 12,000 feet with blockage of the eustachian tube during descent and complete inability to ventilate the middle ear. H. D. S., aged 24.

the 1024 area and to a lesser degree the 5792 area. This pilot has a slight defect in his ability to distinguish certain spoken words and numbers. Repeated tests over a period of two years showed no change in this pilot's hearing Ten consecutive student pilots were tested after training flights of one hour in an open cockpit ship powered with a 240 horsepower motor. In each of these 10 students there was a noticeable drop in hearing in the 4096 area. They were then tested after a four hour flight in a cabin ship powered with a 260 horsepower motor during which they used head phones and a tight-fitting helmet. At the conclusion of this flight, only 3 students showed any loss of hearing in the 4096 area, and this amounted to only 10 decibels. These two series were subjected to practically the same horsepower and noise factor but the second series had the protection afforded by modern cabin soundproofing and the use of head phones and a well fitted helmet. This is proof that a definite loss of hearing follows excessive stimulation by the noise of aircraft unless some form of protection is provided.

AVIATION PRESSURE DEAFNESS OR ACUTE AERO-OTITIS MEDIA

Armstrong and Heim [15] made the first really careful study of the effect of changes in atmospheric pressure on the ears in flight. They studied a group of healthy men in the pressure chamber at various atmospheric pressures from sea level to 40,000 feet (12 kilometers). Their study demonstrated that as the pressure was decreased there was a sensation of fulness in the ear, with bulging of the tympanic membrane, until, at a change in pressure of 15 mm. of mercury, a sudden click was heard by the subject, and the tympanic membrane returned to its normal position. The membrane had been forced outward by excess pressure in the tympanic cavity, and the pressure in the ear was relieved by ventilation into the nasopharynx. In other words, the pressure differential between the middle ear and the external atmosphere had been equalized through the eustachian tube. As the pressure was decreased still further, the click was heard at about each decrease of 11.4 mm. of mercury, or about once in each 435 feet of ascent, of barometric pressure. When air pressure was increased as in descent, an entirely different effect was noticed. The symptom of fulness was the same, but the tympanic membrane was seen to be retracted. At a negative pressure of about 60 mm. of mercury the pain in the ear was severe and resembled that of acute otitis media. The tinnitus was marked, and there was usually vertigo with beginning nausea. At a negative pressure of from 60 to 80 mm. of mercury the pain was intense, deafness was marked, and collapse and general shock sometimes followed. The pain quickly subsided, but a dull ache might persist from twenty-four to forty-eight hours. Hearing was diminished, and

15. Armstrong, H. G.: Principles and Practice of Aviation Medicine, Baltimore, Williams & Wilkins Company, 1939. Armstrong and Heim.[10]

vertigo and nausea might persist for some time. Aviators with normal eustachian tubes can usually equalize positive or negative pressure differentials in the tympanic cavity except when a negative pressure of 80 to 90 mm. of mercury or more has developed; it is then impossible for the eustachian muscles to overcome the pressure which holds the fibrocartilaginous portion of the tube tightly closed and it is necessary to decrease atmospheric pressure below that point before the middle ear can be voluntarily ventilated. These tests were done on subjects who voluntarily kept their eustachian tubes closed.

This clinical entity has been referred to in the literature of the United States as "aviator's or aviation ear." In Germany the terms "barotrauma" and "tonetrauma" have been suggested. Armstrong and Heim [10] suggested the term "aero-otitis media." In England, McGibbon [16] termed the condition, "aviation pressure deafness" and stated that he is convinced that some degree of deafness is a constant symptom of the lesion. McGibbon stated that 26 per cent of the total number of aircrew personnel referred to the ear, nose and throat department of his hospital had complained of aural symptoms that arose during loss of altitude in aircraft. This clinical picture occurs only in those who do not properly ventilate their middle ears. Failure to ventilate the middle ears may be due either to inability to open the tube or simply to failure on the part of the patient to do so. Normally the eustachian tube is opened by swallowing or yawning movements. But in some persons when there is inflammation in the region of the eustachian orifice, as from a common cold, infection of the sinuses, tonsils or lymphoid tissue in the nasopharynx, tumors of the surrounding tissue or malposition of the lower jaw,[17] it is impossible to open the eustachian tube and ventilate the middle ear. This condition may be very serious because of the severe pain produced. Occasionally nausea and vertigo are so severe as to render a pilot incapable of handling his ship. There is considerable loss of hearing as a result of these changes, and Campbell [18] showed that these changes occur in the conversational tone range. That is in the 128 to 1024 cycle areas. This is definitely conduction deafness and is due to a limitation of motion of the tympanic membrane and ossicles. If the middle ear is not ventilated soon after the occurrence of such phenomena, serum and sometimes blood pass from the vessels into the middle ear and cells of the mastoid process. This results in a damping effect on the tympanic membrane and the ossicles. There is no definite inflammation; there is simply a reaction to negative pressure with pouring out of serum and blood so as to neutralize the marked negative pressure in the middle ear.

If insults such as these are repeated and the middle ear becomes repeatedly filled with serum and blood, the tympanic membrane becomes greatly retracted toward the middle wall and there are damage of the tympanic membrane and ossicles and thickening of the mucous membrane lining the tympanic cavity. The lining membrane of the eustachian tube may become so thick as to prevent ventllation by any of the usual methods. In some instances a distinct rupture of the tympanic membrane may occur, with complicating chronic otitis media.[18] These changes are of a definitely chronic nature and inhibit the ability of the tympanic membrane and ossicles to properly transmit sound waves to the inner ear. In such cases a chronic conduction deafness results. There is a decrease in ability to hear sound in the normal speech frequencies, but usually the higher tones are transmitted quite well. Audiogram 4 was taken on a young cadet who had been on a practice bombing mission at 12,000 feet (3.5 kilometers) above sea level. On descent he noticed that he was unable to ventilate his left ear. There resulted rather severe pain, and they ascended back to 6,000 feet (1.8 kilometers) before landing, but still he was unable to ventilate his middle ear. He came to my office approximately sixteen hours after the onset. Examination at that time showed the left tympanic membrane to be dull and about in the normal position. The tympanic membrane appeared to be blood stained but was fairly transparent. The middle ear seemed to be filled with a bloody fluid. There was no elevation of temperature, but there remained a slight amount of pain, and the patient complained especially of sound seeming to be far off and indistinct. One notes the marked loss of hearing in the lower tones, while the high tones were still heard readily. A total of 6 such cases have been seen in the clinic with which I am associated in the past four years. In each case there

16. McGibbon, J. E. G.: Aviation Pressure Deafness, J. Laryng. & Otol. **57**:14-22 (Jan.) 1942.

17. Costen, J. B.: Syndrome of Ear and Sinus Symptoms Dependent upon Disturbed Function of the Temporomandibular Joint, Ann. Otol., Rhin. & Laryng. **43**:1-15 (March) 1934. Wilhelmy, G. E.: Ear Symptoms Incidental to Sudden Altitude Changes and the Factor of Overclosure of Mandible: Preliminary Report, U. S. Nav. M. Bull. **34**:533-541 (Oct.) 1936.

18. Campbell, P. A.: Otolaryngological Problems of Aviation in World War II, Ann. Otol., Rhin. & Laryng. **51**:293-300 (June) 1942; The Effect of Flight upon Hearing, J. Aviation Med. **13**:56-61 (March) 1942. Campbell and Hargreaves.[8]

was a marked loss of hearing averaging between 20 and 30 decibels in the 128, 256 and 512 areas. In each case the loss of hearing was temporary, and with release of blockage in the eustachian tube and absorption of the transudate in the middle ear, the hearing returned to normal.

Another form of aviation pressure deafness results from a disturbance of the equilibrium in the mixture of gases in the middle ear and those in circulation in the capillary lining of the cavity.[19] This condition is sometimes noted following descents from high altitudes, during which pure oxygen has been breathed. The gas introduced has a partial pressure in excess of that in the capillaries and will be absorbed in the course of time. This produces a reduction of total volume and a greater pressure from the outside of the tympanum, which necessitates the introduction of more gas. Behnke [19] described cases in which oxygen at a high concentration has been absorbed during sleep following such descent, producing complete filling of the middle ear by serum and blood. He recommends that only ambient air of 21 per cent oxygen content be introduced during the last stages of descent from 10,000 feet. It is quite common for passengers in commercial airliners to complain that the pressure will not equalize for several hours after descent. This is because oxygen at a concentration of 21 per cent must be absorbed down to a concentration of 12 to 15 per cent in the capillaries. Happily, there is a simple solution which requires little attention. If the air introduced is alveolar air, it will approximate closely the composition of that in the capillary. Thus, if the ear is inflated at the end of expiration, following moderate breath holding, the period of recovery will be materially shortened.[18]

TREATMENT

Aviation perception deafness is best treated by prevention. As previously stated, the best means of protection against noise is found to be modern cabin soundproofing, a tight-fitting helmet or a radio telephone head set. Once the hearing loss is established, little can be done to relieve it. Aviation pressure deafness can be largely controlled by a proper selection of flying personnel and proper instruction in the technic of ventilating the eustachian tubes. Persons with either acute or chronic disease of the upper respiratory tract, and especially of the nasopharynx, should be cautioned against flying. The average person swallows involuntarily about every sixty to seventy-five seconds, and it can be seen that a rate of climb or descent of 200 feet (61 meters) per minute will usually cause no discomfort. A rate of 500 to 1,000 feet (150 to 305 meters) per minute will produce only moderate discomfort even though no effort is made to ventilate the tympanic cavities. More rapid descent may catch the subject unaware and create a tympanic vacuum with the trend of symptoms just described. The allowable rate of ascent and descent of commercial airlines is set by the United States Department of Commerce at 300 feet (91.5 meters) per minute, and some companies limit themselves to 200 feet per minute. Furthermore, stewardesses are instructed to awaken patients who are sleeping when descent is started, in order that they may properly ventilate their eustachian tubes. Probably the simplest maneuver to ventilate the normal eustachian tube is to swallow. It may also be accomplished by yawning, by singing, by shouting or by holding the nose and increasing the pressure in the nasopharynx by forcible blowing. Once the eustachian tube becomes blocked and the subject is unable to open it, active treatment must be instituted in order to bring about relief. There are marked loss of hearing and usually great discomfort. Irrigation of the external canal with copious quantities of water at 110 to 115 F. or application of dry heat will give considerable relief. Shrinkage of the eustachian tube with gentle politzerization or catheterization of the tube is indicated to relieve the temporary vacuum in the middle ear. Shea, in a personal communication, recommended puncture of the tympanic membrane in the lower anterior quadrant, near the eustachian orifice, with a hollow needle, to reduce this partial vacuum in the middle ear, when it is impossible to ventilate the middle ear properly through the eustachian tube. Any acute or chronic infection or other abnormality of the nasopharynx should also receive appropriate treatment.

CONCLUSIONS

1. Loud aviation noise produces acute fatigue of the end organ of hearing, which results in a definite loss of hearing in the frequency region of 4096 cycles per second.

2. If exposure to excessive noises is allowed to continue, chronic accumulative fatigue of the end organ of hearing results.

3. All pilots are not affected to the same degree.

4. The hearing loss in aviators is not unlike that in other persons who are constantly exposed to noises of great intensity.

19. Behnke, A. R., and Willmon, T. L.: Physiological Effects of High Altitude, U. S. Nav. M. Bull. 39:163-178 (April) 1941.

5. Hearing losses of this type are frequently not detected since the speech area is not involved.

6. Aviation pressure deafness is a result of failure or inabilitiy to ventilate the tympanic cavity properly on descent.

7. It is more frequently found in inexperienced flyers or those with some disease of the nasopharynx and eustachian tube.

8. Marked loss of hearing in the conversational areas is a constant finding.

9. The hearing loss is usually temporary and is restored on restoration of the proper pressure equilibrium in the middle ear.

10. Repeated blockage of the eustachian tube with resulting injury to the middle ear may result in permanent changes in the middle ear with permanent loss of hearing of some degree in the conversational areas.

11. Modern cabin soundproofing, the wearing of a proper helmet and well fitting ear phones are doing much to eliminate hearing defects secondary to excessive noise. Regulation of ascent and descent in commercial airlines, together with proper selection of personnel and instruction of passengers, is making aviation pressure deafness much less common than in the past.

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THE AGING EAR

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The falling birth rate, the relative decrease in the number of children under 5 and the doubling of the number of persons over 65 have caused a shift of population into the older brackets, and the indications are that this shift will continue. The otologist as well as the physician in general practice will therefore be confronted with more aging people and more aging ears than in the past. The great difficulty in gerontology is to determine which processes, if any, are physiologic and which are pathologic and how these two interact in causing what for lack of a better name we call "normal aging" and "premature aging."

There has been much speculation as to the fundamental cause of aging, with few convincing conclusions, owing largely to the fact that every living thing inherits limitations, deformities or constitutional weaknesses and is exposed to inevitable detrimental influences during life, and these have an accumulative effect with the years. Some effects may be mitigated, but only incidentally, by homeostatic control and by the influence of environment, diet and vascular, hormonal, metabolic and nervous factors.

Whereas biochemical and structural changes ultimately determine function, in most tissues they are slow and hence difficult to detect and measure at short intervals with delicacy and precision. Considerable variations may occur, become stabilized and cause but little or no change in function for long periods. The body as a whole ages because of loss of function, not simply because this or that tissue or this or that organ is out of normal order, missing or destroyed. Readjustments and compensations may within certain limits adequately prevent crippling effects for a long time and so permit a continuation of function sufficient for the senile needs.

Aging is a progressive irreversible downward trend in function, and the auditory mechanism is perhaps better adapted for the detection of beginning and later slight changes in function than any other organ in the human body. Its end organ is a highly protected and specialized tactile apparatus, deep set in a housing composed of the densest bone in the body (the petrous bone). Except for incapacity to hear warning sounds or the approach of danger, animate or inanimate, imperfect hearing alone is not a cause of death, and comparatively few diseases of the ear are lethal to the ear or to the sufferer.

Even before birth the otic capsule (including the stapes) attains adult dimensions, configuration and histologic structure while other parts of the body are still in process of fetal development, so that it may be said to be fully grown but not yet of this world. Soon thereafter the ear functions at its highest efficiency and from then on slowly declines. Though it reaches maturity twenty odd years before the body as a whole, it appears to inherit a functional expectancy terminated only by death of the body itself.

The otic capsule reacts to disorders of the endocrine glands and of metabolism in unique ways. Its enchondral layer (intrachondral [Bast]) is the only bone in the body which normally persists throughout life without change. With the other layers it surrounds a closed endolymphatic system and an end organ with a vascular supply which is terminal. Even after death the cochlear electronics persist for a while. These facts, together with the spatial arrangement of the auditory neural elements and the easily applied means for testing them, make it possible to measure any slow functional decline over a longer time and with an accuracy and dispatch unattainable with any other organ of the body.

The human external ear is of comparatively little functional importance but, like other appendages of the body, shows changes with age. These changes, beginning in middle life (30 to 50), are: atrophy; loss of elasticity; dehydration; pigmentation; growth of single long hairs from most unexpected locations, especially along the periphery of the helix and on the tragus; telltale wrinkling in front of the tragus, especially noticeable in women after the menopause, and in men a flabbiness and lateral prominence oftentimes ludicrous to behold. The drum membrane and the skin of the meatus often show a tendency toward, or even marked, thinning, and in the external meatus, which may be enlarged from atrophy of its walls or narrowed from bulging thereof, a stiff jungle growth of hair which entangles cerumen, sweat, exfoliated epithelium, soap and dust from the environment,

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so that impaction of the mass in the fundus is common and removal often most disagreeable.

When the drum membrane is sufficiently thinned, one can see the outline of the long process of the incus, the articulation of the stapes and the malleus and frequently the crura of the stapes. Unless there is a loss of tension, this atrophy alone does not appear to diminish the hearing.

The eustachian tubes in aged persons when not diseased are usually more patent than those in young persons, and the muscles, tendons and ossicles of the middle ear probably atrophy to some extent, but this is at present largely a surmise. The mucous membranes generally are less succulent and the lymphoid tissue less evident. The impression obtained is that atrophy or involutionary processes are responsible for most of these conditions.

All aging processes are accentuated by cardiovascular insufficiencies, but these are spotty and do not parallel the loss of function or the degree of atrophy; they constitute disease and not "physiologic aging." Extremely old persons, to have survived, must have been free from prolonged or severe vascular changes. Their chronologic age may vary greatly from their biologic age.

Guild [1] stated that histologically there is no significant difference between the vestibular structures from young and old people, but even microscopic measurements are crude compared with the ultramicroscopic changes which affect function. There is no question but that after 35 to 40 every one shows signs of aging, such as delayed reaction, diminished security of orientation and equilibrium under sudden or unexpected changes in position of the body in space and lessened muscular coordination (or slowing down) in adjustment to such episodes.

The vestibular apparatus of the ear is much older phylogenetically than the cochlear mechanism, but what effect this has on its biologic age in a given animal is not clear. All tests now employed are too crude to be used for measuring its gradual changes during aging. Moreover, training, occupation and many psychosomatic factors make measurement of vestibular capacity uncertain. In none of the labyrinthine tests are accurate threshold stimuli employed, and therefore all engender a "recruitment" effect (Fowler), which is the universal response of all sensory mechanisms to over threshold stimulations. This effect gives the impression of a near normal response, although the threshold is elevated considerably. Although there are no tests for small changes in vestibular function, audiometric measurements provide them for hearing acuity.

The auditory neural mechanism is still a mystery. Reasoning by analogy, all of its tissues should undergo atrophy or degeneration with aging, but the only impressive histologic data concern the degeneration of the neural elements (nerve fibrils and nuclei [1] of the spiral ganglion). But similar changes are seen in ears obtained from young people, resulting from various insults, heredity and vascular episodes, even in those ears in which there appears to have been no deafness for any tones. The cochlear atrophy does not closely parallel in amount or selectivity the functional changes (Guild).[1]

It has been known for centuries that people grow deafer as they grow older, but graphs such as those published by Bunch and Raiford [2] and Ciocco [3] show that in succeeding decades (3 to 7 inclusive) the progressive loss in capacity varies with age and with frequency (for air conduction). Males show a somewhat greater loss than females. My own determinations for both air and bone conduction (decades 3 to 8) show similar trends in people with cardiovascular disease.[4] The frequency region near 4000 cycles per second air conduction appears on the average to be affected more than the frequency areas above and below it.

To call these impairments normal for the different age groups is customary but misleading, because they are not normal losses; they are deviations from what is called no loss (the average normal or zero). There is no such thing as a normal loss at any age, but one may determine the average deviation at any age. The reference standard for any age or decade should be called the average deviation from normal for such age or decade. To call anything normal which is below the average of the best is, without qualification, misleading. There are too many variables in the averages obtained from different people in subnormal groups to make those averages dependable as a standard of reference. The acoustic impairments of old people may vary from losses of almost nothing to total deafness, and yet, just because they are old, both those

1. Guild, S. R., in Cowdry, E. V.: The Problems of Aging, Baltimore, Williams & Wilkins Company, 1942.

2. Bunch, C. C., and Raiford, T. S.: Race and Sex Variations in Auditory Acuity, Arch. Otolaryng. **13**: 423 (March) 1931. Bunch, C. C.: Further Observations on Age Variations in Auditory Acuity, ibid. **13**: 170 (Feb.) 1931; Age Variations in Auditory Acuity, ibid. **9**:625 (June) 1929.

3. Ciocco, A.: Observations on Hearing of 1,980 Individuals: A Biometric Study, Laryngoscope **42**:837 (Nov.) 1932.

4. Fowler, E. P.: The Effect of Severe Illness upon the Hearing, Ann. Otol., Rhin. & Laryng. **43**:387 (June) 1934.

with good and those with bad hearing have been thrown together to attain what is called an "average normal" for the age group. It would be almost as logical to use the right and the left ear of an old man who has nearly normal hearing in one ear and a loss of 60 decibels in the other and call this man's hearing the average normal hearing for his age. I must again point out that neither for air conduction nor for bone conduction do threshold audiograms reveal the whole story of functional capacity. Above threshold graphs are needed to clarify the picture (Fowler).

In many old people, although the hearing is excellent for all but some of the high tones, the hearing for speech, particularly in a crowd, is distinctly affected. Whether this is due to loss of some of the higher speech frequencies which contain the overtones contributing much to the characteristics of individual voices[1] (such losses being due to aging or degeneration in the peripheral, central or cortical tracts, in their nuclei, myelin sheaths or intercellular tissues) or to psychologic factors, I do not know. I suspect it may be due to both.

It is interesting to note that the reflexes, the vibratory sense and all tactile sensations diminish in old people. The loss of vibratory sense may in some way account in part for a loss in bone conduction more than for one in air conduction, as sometimes recorded. If bone conduction is a true test for nerve function, I cannot otherwise explain how it can ever be less than air conduction except there be interference with the escapement in both labyrinthine windows.

Osteoporosis in aged persons is due to normally rapid resorption, on one hand, and deficient deposition of calcium on the other; failure to lay down a matrix or to deposit calcium in the matrix occasions osteoporosis and osteomalacia, respectively. Osteomalacia is really an adult form of rickets. Osteoporosis may occur at any age, and conditions resembling it may follow hyperparathyroidism and chronic disabilities.

The petrosa, like other parts of the temporal bone, in age may show evidences of hyperostosis, but patchy or widespread atrophy is usually not present. The meninges overlying it tend to thicken and become adherent and may show calcification. In the pons Varolii and the medulla oblongata the nerve cells become reduced in number and many show atrophy. Surviving neurons become smaller and reduced in number. There is a widespread increase of pigment and changes in the intracellular fibrils. When the cell bodies later disappear, there remains a skeleton of tangled coils, especially noticeable in the allocortex of the temporal lobes and proliferative as well as regressive changes in the neuroglia are also seen.[5] Just outside the nerve cells and especially around the blood vessels in certain parts corpora amylacea and depositions occur. Iron deposits in perivascular spaces and "senile plaques," which are argentophil formations, are scattered in the cortex, especially in the deeper layers (occipital, frontal and temporal) and even in the brain stem (not pathognomonic of senility). Involutionary changes in blood vessels and arteriosclerosis occur, but these may not be interdependent. Physiologic and pathologic processes are simultaneously operating, though not necessarily cooperating, in the production or the retardation of the involutionary processes, and their interrelationship is undetermined. Cytologic study of the anterior lobe of the pituitary gland in fowl led Payne[6] to the conclusion that in very old fowl this gland was probably not functioning.

Myer (1920) observed calcification and hyalinization in the basilar membrane of the basal turn of the cochlea in 28 people over 60 years of age, but these changes are also observed in younger people.[1] Truex and Zwemer[7] described fatty degeneration in sensory neurons which leads to complete destruction of some cells and their processes. This wholesale destruction after the fourth decade may be correlated with a clinical loss of vibratory sensitivity in corresponding age groups in man. Similar changes have been observed in autonomic ganglions (Kuntz). The over-all picture strongly suggests gradually progressing anoxia. In addition to the deafness, other symptoms such as tinnitus and vertigo, commonly occur and tie in with this suggestion. It is not known why sensory cells are so extensively involved while the sympathetic and other motor neurons largely escape this degenerative process.[7]

Changes similar to those enumerated may be surmised to occur in the central auditory tracts, but few investigations have been instituted along these lines. It is important that all otologic research laboratories look into these matters.

The audiograms of old people frequently remain stable for many years. Does this mean that the aging ear has ceased to age or that the deafness observed in old age is due to diseases which have ceased to progress? I have in my

5. Critchley, M., in Cowdry, E. V.: The Problems of Aging, Baltimore, Williams & Wilkins Company, 1942.

6. Payne, F.: The Cytology of the Anterior Pituitary of the Fowl, Biol. Bull. **82**:79 (Feb.) 1942.

7. Truex, R. C., and Zwemer, R. L.: True Fatty Degeneration in Sensory Neurons of the Aged, Arch. Neurol. & Psychiat. **48**:988 (Dec.) 1942.

files audiograms taken over a considerable length of time. I show here tests of 1 person taken several times a year from the age of 48 to the age of 71 inclusive. To conserve space only 13 are shown (table). It will be noted that there are changes of as much as 25 decibels up and down during this period and a distinct tendency toward a drop in the upper frequencies as aging progresses (both for air condition and for bone conduction). In this person there were no systemic diseases or any disorders of the ear except once, when with a head cold and asleep the patient made a rapid descent in a stratoliner. There was then a sudden pain with blockage of the eustachian tube in one ear until the ship again ascended. In middle life (from 19 to 45 years of age) there were several acoustic shocks with deafness for all sounds in the left ear, at one time almost total for as long as fifteen or twenty minutes, with rapid recovery. has been little or no change in the hearing for the middle and lower tones (no signs of aging). Are these progressive losses in the high tones due to physiologic aging or to the internal and external environmental insults suffered during life? I do not know.

Hearing Acuity as Shown by the Audiograms of One Person Between the Ages of 48 and 71 Inclusive (Twenty-Three Years)

Year	128	256	512	1024	2048	4096	8192	9747	16384	Audiometer	128	256	512	1024	2048	4096	8192	9747	16384	Comment
1921	5	0	0	0	5	10	10	..	10	1-A	−10	−10	−10	−10	10	5	25	..	20	
1924	0	5	5	0	5	5	15	..	15	1-A	0	−5	−5	−10	10	5	30	..	25	
1927	5	5	0	−5	−5	0	5	30	..	1-A	0	−5	−5	−10	15	10	5	35	..	
1930	5	0	0	−5	5	5	10	30	20	1-A	0	0	0	−5	15	15	10	35	30	Emotional strain
1932	5	5	0	0	5	10	10	30	20	1-A	0	0	0	0	15	20	15	35	..	Emotional strain
1934	0	5	−5	0	10	5	5	..	20	1-A	0	−5	−5	−10	20	25	15	..	40	Tinnitus in left ear
1936	0	0	10	5	15	15	30	30	..	1-A	0	−5	0	−5	20	25	30	40	..	
1937	5	10	5	5	15	20	30	20	..	1-A	0	5	15	15	15	20	30	..	..	Tinnitus in left ear
1938	0	5	5	15	10	35	35	..	..	1-A	5	0	0	−5	0	35	10	..	..	Tinnitus in left ear
1939	−10	0	−5	−5	−5	0	20	..	..	1-A	−5	−5	−5	0	0	5	30	..	..	Tinnitus in left ear
1940	0	0	0	5	5	10	35	30	..	6-B	0	5	−5	−5	0	10	30	25	..	Tinnitus in left ear
1941	−5	0	0	0	−10	0	20	..	..	6-B	0	0	0	0	0	0	35	..	..	Tinnitus in left ear, high frequency
1944	5	5	5	0	5	15	25	30	..	6-B	10	10	5	0	5	30	35	45	..	No tinnitus; slight cold
Bone conduction		0	−10	−10	0	15	15	30	..	...	..	0	−5	−10	10	30	30	45	..	

Practically every few days for a large part of each year indoor and outdoor rifle and pistol firing were indulged in. Several attacks of malaria in youth and one in middle age necessitated the taking of 20 to 30 grains (1.29 to 1.94 Gm.) of quinine. In middle age this or something else brought on an intermittent but sometimes a severe over-all frequency tinnitus. After the age of 50 a high-pitched tinnitus appeared off and on in the left ear, particularly after excessive exercise, smoking or consumption of alcoholic beverages. A high frequency tinnitus is now present, but this recedes or ceases for long periods without apparent cause.

It may be that some of the wider variations in the audiograms were caused by colds, fatigue or changes in environmental noise, but for the past twelve years all tests were done in a soundproofed room, and it was during these years that the progressive losses for the high tones became evident. There is no difficulty in hearing speech except in the back of large auditoriums (without microphones). It will be noted that there

DISORDERS AND DISEASES OF THE AGING EAR

It may be argued that aging though inevitable is in itself a disease, although its pathogenicity is controversial. If it results from repeated trauma, intoxication, anoxia, and other injuries it should be largely preventable, but if it is due wholly to the passing of time, nothing can permanently stop it. But time alone not infrequently reveals no deafness.

Aural diseases in old people often show atypical features and little if any correlation with most of the systemic diseases. They are more asymptomatic and multiple and less pathognomonic. Cardiovascular insufficiencies usually add to or accentuate many diseases of the ears in old people.[4] The senile ear, like the senile body, is a kaleidoscope of pathology, but the tendency in diagnosis is to focus on a single lesion. The prognosis is usually unfavorable because the damage is often irreparable. Infections tend to be either much more severe or much less severe than in earlier years. Immediately after rather than during a crisis may be the most precarious period so far as function is concerned.

The ear is subject to the same diseases in old age as in youth, but it is usually free from the infectious diseases of childhood and less exposed to damage from drugs, poisons, traumas, explosions and swimming. Although the eustachian tubes seem to be more patent in old than in young persons, the cilia, like other specialized epithelial elements may be less responsive to the presence of obstructing secretions and less effective in their removal.

In my experience, acute infections in the middle ear and its contiguous cavities have tended to be less fulminating but slower in resolution than in young patients. So-called aero-otitis, though less often encountered in old than in young people, is often stubborn to treatment. Spontaneous pain is usually less and sometimes absent even in severe bilateral acute mastoiditis. The healing of the integument and wound cavity after mastoidectomy is slower but less interfered with by excessive granulations. Some excellent functional results following radical mastoidectomies have been obtained in older people.

The frequency of aural disorders decreases in middle age, but in elderly persons there is a noticeable increase, not only because of nerve deafness from atrophy of Corti's organ (or the central neurons) but also in some instances because of insidious development of chronic productive inflammation of the middle ear resulting in obstructive deafness.

Recurrent dizziness is quite frequently encountered in people over 50 and is often associated with deafness and tinnitus (so-called Ménière's symptom complex). All of these symptoms commonly derive from disorders of the neural mechanism, in large part from vascular changes which in younger people may cause transient, few or no disturbing symptoms. The neural mechanism of the aged ear seems unable to endure insults which in youth are hardly noticeable.

Tinnitus is frequent in old people. It most often resembles seething or escaping steam or hissing (like a thermal noise) with occasional thumping or ringing. Of my elderly patients, over 90 per cent have experienced a "nonvibratory tinnitus."[4] The frequency band of tinnitus in the aged is almost always within the frequency band of the depressed higher frequencies, and in my opinion the tinnitus and the deafness are in most instances caused by similar or identical lesions. The tinnitus is more often difficult to mask with the thermal noise or similar sounds than in youth. That local anoxia plays an important part in both the loss in function and in the production of the tinnitus seems probable. In any event when the circulation is improved and the tinnitus diminishes or ceases, the hearing is better, and herein lies one of the great services the otologist can contribute to the aging patient.

I have set forth elsewhere my procedures and philosophy of treatment for people with tinnitus, and I shall not therefore give it space at this time. I should, however, like to make it plain that I believe the otologist should not lose interest in the elderly patient with or without tinnitus just because he knows he cannot restore the hearing. Almost without exception the older patient can be helped in many ways.

One often hears it stated or implied that certain local infections, e. g., sinusitis, an abscessed tooth or an infected tonsil, cause deafness. But the remarkable thing is that, like the systemic infectious or noninfectious diseases, they so often do not appear to affect the function of hearing. Aside from its effect on suppuration, diabetes has not been correlated with the deafness of aged persons. Extreme degrees of anemias and leukemias occur in old people with little deviation from the average best in hearing function. However, cardiovascular-renal disease is a real threat because the changes it causes throughout the body, though spotty, seldom in the long run entirely avoid damaging the fluid or solid tissues in some part of the neural mechanism of the ear.

Acute and subacute nonpurulent otitis media (mucositis) is common, but chronic otitis media serosa is uncommon. So-called dry catarrh, better called otitis media sicca, is common and is damaging to the conduction apparatus in proportion to the productive inflammations engendered. The mucosa of the eustachian tube is also usually involved. Irrigations with hot isotonic solution of sodium chloride or use of heat in any form as well as radon irradiations aid in controlling lymphoid hypertrophy, exudation and any accompanying otolgia or neuralgia.

There may be some general or special bodily disorder to combat. All foci of infection should be cleared up. The general health often needs attention more than the ear.

Otosclerosis may or may not progress with age, but clinically the impression is that it always does so when the loss of hearing is progressive. If the otologist will examine his charts carefully, he will see that obstructive deafness from otosclerosis often does not increase in aging but that most of the progressive deafness is due to increasing loss from neural lesions. In other words, clinically the otosclerosis has remained almost or quite stationary, while the nerve deafness has increased as is common in old age. However, the nerve deafness also may remain stabilized many years.

Guild's latest statistics[8] reveal 13 people with histologic otosclerosis who died when over 50 years of age. The lesion was anterior to the oval window in one ear in 12 of these people, but not 1 showed any ankylosis of the stapes, and only in 3 was the process active. Three of the 13 showed areas near the round window (in the right or the left ear) but in only 1 of these was the process active.

8. Guild, S. R.: Personal communication to the author.

In 113 cases with otosclerosis, according to Guild,[8] without subdivision of the areas according to location (oval window, round window) but with grouping only by twenty year periods, the division into active (A) and quiescent (Q) areas is: under 20, 15 A and 2 Q; 20 to 39, 14 A and 15 Q; 40 to 59, 27 A and 22 Q; 60 plus, 2 A and 16 Q.[8]

These histologic observations confirm my clinical observations that otosclerosis in aged persons is usually inactive (not progressive). To a certain extent it may be said to progress with chronologic age but it cannot be said to progress because of it or, for that matter, with biologic age. It would appear to have no correlation with age as such but considerable correlation with certain periods of biologic age (i. e., puberty, childbirth, menopause). All of this is most interesting from the standpoint of causation, prevention and treatment.

Heredity and constitutional factors are of prime importance in the ability to overcome disease in the aging ear as well as in the body at large, but hygiene, environment and diet play an important part. I have not been impressed by the vitamin treatment for deafness in people over 40 although practically all of my patients are "full up" with vitamins. In some, severe reactions developed from them.

Old people are prone to lose faith in doctors. They want too much and have received too little. They are difficult to manage and like to give orders, not receive them. Defeatist attitudes in both patient and physician are largely responsible for the despair and suffering of the old.

More moderation and conservatism in treatment is indicated than in the young person. At least the life of the patient as well as the life of the ear should be prolonged by not shortening it through damaging treatment. Slight disorders should be investigated seriously. Early detection is important as well as an annual checkup to forestall avoidable disabilities. Education in preventive measures, and social adjustments are indicated. Lip reading and hearing aids when necessary must be prescribed and accepted with understanding. The societies for the hard of hearing give invaluable help along these lines. Old men are even more inapt (or more unwilling) than old women in learning lip reading and in accommodating themselves to the use of hearing aids.

Hard of hearing people should not retire from the hearing world. Their work and play should and can be adjusted sufficiently early to anticipate rather than follow the shock of incapacitating deafness. The science of otologic gerontology will, I believe, make great progress in the years to come, but the outlook for indefinitely avoiding aging processes in any organ seems at present hopeless.

It is apparent that this over-all picture which I have painted of aging reveals, almost alone, the pathologic side, but it may be there is no aging without pathologic change. I incline to this possibility which leaves the door open for treatment. But in any case, if every one is prepared to meet the inevitable without undue resentment, with some dignity and with a sense of humor, their old ears, hear they well or hear they poorly, will not take all the joy out of life.

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SULFONAMIDE COMPOUNDS IN THE TREATMENT OF INFECTIONS OF THE NASAL SINUSES

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The use of the new bacteriostatic agents to control infections of the nasal sinuses has been taken up with high hopes by both physician and patient because of the spectacular results obtained by their use for the control of infections in other parts of the body. The physician, in his eagerness to offer something for the treatment of sinus infections that would be better, or at least more rational, than nose drops, oral vaccines, inoculations and short wave diathermy, welcomed the advent of the new drugs with unrestrained enthusiasm. It is now three years since the first favorable report was published on the curing of sinus infections by spraying a solution of a sulfonamide compound into the nose. Rhinologists now should be in a position to take stock and estimate what benefits or damages have resulted from this mode of treatment that three years ago was hailed as a cure for sinus disease.

In the approach to the problem of nasal sinus disease, full value must be given to the importance of the common cold, a virus infection, self limited in its duration and recognized by all as a head cold with sneezing, congestion of the nose and excessive secretion of nasal mucus.

A bacteriologic study will show that in most noses there are present at all times potentially pathogenic bacteria. From the nose of a person in apparent good health there can be recovered streptococci, staphylococci, pneumococci and Haemophilus influenzae along with less common bacteria. Some or many of these organisms are perennial in the nasal cavities of many people. With the invasion of the nose or the nasopharynx by the virus of the common cold, these bacteria at once take on great activity, with increase in numbers and virulence. In the cold sequence this is the important but unexplained phase causing the complications attendant on or following acute coryza. It is probable that the presence of the cold virus in some way activates the normal bacteria of the nose, and with the increase in growth and virulence of these organisms one observes the expected extension of the infection to the nasal sinuses, the middle ear, the bronchial tree or even the blood stream. It is at this point in the progress of the infection that bacteria in great numbers can be recovered from the nose and the nasopharynx. There may be a mixed growth of organisms or, what is more usual, a predominant growth of a single type: streptococci, staphylococci or H. influenzae.

It is evident that if the common cold or coryza could be prevented or controlled, there would be no succeeding infection of the nasal sinuses by the impotent bacteria that dwell in the nasal cavities. While the cold virus remains unknown, the problem of finding a specific substance to combat it is a difficult one. Since there is no specific treatment for the virus of the common cold, one's effort must be directed against the complicating bacterial infection of those structures that are extensions of the nasal cavity. Certain basic principles of treatment will hold good whether the sinus infection is the fulminating sinusitis seen in young people, the acute sinus infection following a head cold or the long-continued sinus infection with acute exacerbations.

Because of the marked success attending the local use of the sulfonamide compounds in open wounds and surgical cavities, rhinologists seized on these drugs for local application to the nose in cases of infection of the nasal sinuses, overlooking for the moment the fact that the unbroken mucus membrane of the nose and sinuses does not offer the same favorable setting for the action of these compounds as does the raw surface of a wound produced by trauma or surgical operation. It is apparent that their enthusiasm outran their sober judgment, and as a result the drug market was flooded with drops, sprays and powders to be instilled, sprayed or insufflated into the nose in the treatment of sinus infections. Many favorable clinical reports were published by reputable observers on the effectiveness of this mode of treatment.

In nasal sinus infections the mucous membrane of the nose and sinuses is edematous, with the result that the airway is limited and the sinuses are changed into closed cavities. Under these conditions, medication introduced into the nose does not enter the sinus cavities and cannot come in contact with the organisms that are the cause of the sinusitis. Nothing will be accomplished by destroying bacteria in the nasal cavity

when the site of the infection is in the sinuses, now closed by the engorgement and edema of the mucous membrane of the nose. It is possible that the favorable results that have been reported on the local use of the sulfonamide compounds in the control of sinus infections have been due to the vasoconstricting drugs that are combined with them in medication. The shrinking of edematous nasal mucous membrane is an effective method of treating nasal sinus disease, and it is difficult to evaluate the respective importance of a sulfonamide compound and a vasoconstricting drug when the two are combined.

Since it was determined that nothing could be accomplished by destroying bacteria in the nose when the infection was in the nasal sinuses, completely shut off by edematous mucous membrane, it was planned to bring the sulfonamide drugs into direct contact with the offending bacteria at the site of the infection. The displacement method of creating a vacuum in the nasal sinuses and by the release of the vacuum allowing medicated fluid to flow from the nose into the sinuses is an accepted method of treatment of sinus disease. It has never been a satisfactory mode of treatment in my hands. Because of the many favorable reports by those who have used this method, an experimental review of this mode of treatment was carried out.

A number of contrast agents were used in the experiments to determine how much of the opaque mediums could be made to enter the sinuses by the displacement method of producing a vacuum in the sinuses. Iodized poppyseed oil, diodrast and a suspension of bismuth were in turn used as contrast mediums and the results checked by roentgen examination. It was observed in this series of experiments with contrast mediums that in most of the subjects the sphenoid sinuses were well outlined. In a moderate number the maxillary sinuses showed some of the contrast medium. In a limited number some of the opaque material could be seen in the ethmoid cells. In none of the roentgenograms was any of the contrast material seen in the frontal sinuses.

I was convinced after this review of the displacement method that no reliance could be placed on this mode of causing medication to enter the sinuses by creating a vacuum in them. Again I think that the favorable results reported from this method of treatment of nasal sinus disease have been due to the preliminary shrinking of the membranes by the vasoconstricting drugs which are made use of in the displacement treatment preliminary to creating a vacuum in the sinuses.

A second approach was now made to the problem of bringing medicaments into contact with the nasal sinuses by way of the nose. Industry has recently made use of active surface compounds having marked wetting and penetrating characteristics. These wetting agents are widely used in the textile industry and in bottling plants, dairies and canneries for cleaning purposes. They are known to have a low degree of toxicity and as used in industry are found effective in concentrations as low as one tenth of 1 per cent. Many of them have marked bactericidal activity. In laboratory animals these wetting agents have been used as vehicles to carry the sulfonamide compounds in an effort to determine the penetrability of the cornea to these drugs. Bellows and Gutmann found that the corneas of laboratory animals showed an increase of penetrability of from two to fifteen times for sulfathiazole when a wetting agent was used as a vehicle to carry the drug.

A wetting agent may be described as a compound which lowers interfacial tension. The compound is made up of molecules containing both polar and nonpolar groups. When applied to an epithelial surface, the molecules concentrate on the surface and arrange themselves so that the polar group is in contact with the epithelium and the nonpolar group is directed out into the surrounding medium. As a result of this arrangement of the molecules, the interfacial tension between epithelium and the surrounding medium is decreased, which results in increased penetration by both the wetting agent and any substance that it carries in solution or suspension.

Numerous wetting agents, now more than one thousand, are available to industry under different trade names, and each has a different chemical structure. Most of them are alcoholic sulfates. Others, like zephiran and ceepryn, are quaternary ammonia compounds. The agents made use of in these experiments were aerosol, tergitol and ocenol.

As a working basis it was planned to use the various sulfonamide drugs in solutions of aerosol and ocenol. The solution was to be sprayed into the nose with the hope that the high degree of penetrability of the wetting agent would enable both agents to penetrate the surface of the infected mucous membrane as well as the ostiums of the sinuses. This study was carried out solely as a clinical experiment, and no attempt was made to exercise laboratory control over the amount of the sulfonamide compound that penetrated the membrane of the nose or the sinuses. My chief interest was to determine whether the wetting agent would be effective as a vehicle in forcing penetration of the membranes and cavities with the suspended sulfonamide drugs. Because no control was possible, these experiments

stand as purely clinical tests without laboratory control.

Many of the wetting agents used were found to have objectionable features that could not be overcome. With some agents the hydrogen ion content could not be brought to the proper point between 5.5 and 6.5, the level that has been found to approximate the reaction of normal nasal secretions. Some of the wetting compounds were found to be incompatible with the sulfonamide compounds, and some were highly irritating to the nasal membranes. The series was finally concluded with the use of a 2.5 per cent suspension of sodium sulfadiazine in aerosol.

A small series of cases of what we considered the common head cold were first studied. This series was limited in number because of the difficulty of finding cases in which the infection was in the stage of onset. Patients complaining of sneezing, congestion of the nose and excessive watery nasal secretion and without a previous history of nasal allergy were thought to qualify for admission to this group. The mixture of aerosol and sulfadiazine in the concentration of 2.5 per cent was sprayed into the nose, three compressions of the hand bulb of the atomizer being used as an arbitrary dose. The spraying was repeated at hourly intervals. This routine was continued for three days, and then a survey was made.

The following observations were made over the three day period: The wetting agent with sulfadiazine did not produce vasoconstriction. None of the disagreeable symptoms, such as sneezing, nasal congestion and nasal discharge, were relieved. The duration of the attack was not cut short. The patients did not express appreciation or gratitude for the treatment. In a few cases there was moderate irritation of the nasal membranes. There were 2 cases in which the disease process went on to a bacterial infection of the middle ear, but the total number of cases was so small that no conclusions could be drawn as to the percentage of bacterial complications.

A number of patients with frank sinusitis were treated by this method with sulfadiazine in aerosol. The routine of hourly spraying was followed out with no satisfaction to either the patient or the physician. In these cases no shrinking of the nasal membranes was observed. The purulent discharge in the nasal cavity and middle meatuses persisted, and the usual symptoms and signs of pain, congestion of mucous membrane and purulent nasal discharge were unchanged. The detergent action of the wetting agents was sought for as manifested by liquefaction of the nasal discharge, but such a detergent action was not observed. The blood levels of sulfadiazine at the end of three days of hourly spraying varied from 1.5 to 2.5 mg. per hundred cubic centimeters.

The promise held out by these wetting agents as vehicles to carry the sulfonamide drugs for local application to the nose as a treatment for nasal sinus infection was not fulfilled. They are remarkable compounds in their action as detergents and as emulsifying and penetrating agents, but they do not penetrate the nasal membranes or the nasal sinuses. At this point one is reminded that it has never been considered rational treatment of pneumonia to instil medication into the trachea because of the presence there of purulent material, the product of infected lung tissue. On the same basis medication of the nasal cavity appears futile for no other reason than that the products of infection are present there while the site of the infection is far removed and sealed off in the sinus cavities.

During the course of these experiments, when it was evident that the local application of the sulfonamide compounds to the nasal cavity for the treatment of sinus infections was unwarranted because of the unsatisfactory clinical results and because of physical and mechanical laws, there appeared a report by Russell Cecil and his associates on their investigation of the treatment of the common cold with sulfadiazine. Cecil observed that the oral use of sulfadiazine did not shorten or alter the course of the common cold. He did note that a bacteriologic study revealed a consistent reduction in the total number of organisms recovered from the upper respiratory tract in cases in which 1 Gm. of sulfadiazine was administered three times daily. He was interested only in the common cold and did not include the bacterial complications attendant on this virus infection. The marked reduction in the number of bacteria in the nose with small doses of sulfadiazine seemed to indicate another angle of approach to the treatment of nasal sinus disease.

The opinion expressed in a recent paper on sinusitis that "we should never resort to surgery as long as adequate progress can be made by conservative treatment" is a concise description of the wave of conservatism in the treatment of sinus infections that swept over the country fifteen years ago. I think this wave did much to submerge what should have been a serious effort to perfect the surgical treatment of sinusitis, infections that have all the characteristics that are recognized as making up an infection calling for surgical treatment. The crest of this wave brought in all the wreckage of conservative treatment: nose drops, spraying solutions and diathermy.

I believe that an infection of the nasal sinuses is a serious malady, especially in young people.

Such infections are prone to return each year or more often and are the cause of repeated and prolonged periods of disability. I find that patients suffering from acute sinus infections dismiss themselves from treatment when symptomatic relief is obtained, long before signs and symptoms of the infection have disappeared. These patients are almost certain to have recurrences of infection, which are only reactivations of the original infection that was never cured.

It is my opinion that an acute infection of the nasal sinuses in any age group requires prompt intensive treatment, which should be continued to the point of complete cure when such a cure is possible. It is for this reason that I make use of the newer chemotherapeutic agents in adequate doses, supplemented by surgical measures when these are indicated. Conservative treatment of sinus infections under my direction has resulted in some relief of disagreeable symptoms but has not prevented recurrent attacks that gave displeasure to both patient and physician. These are some of the reasons I offer for the early use of sulfadiazine in full doses, in the treatment of nasal sinus infections.

Not all infections of the nasal sinuses require treatment with the sulfonamide compounds. One always has in mind that the sulfonamide drugs are highly toxic chemicals. One knows that patients may become sensitized to these drugs, and while toxic symptoms may not appear during a course of treatment, it is possible for them to manifest themselves in a violent manner when these drugs are given for some succeeding illness. The decision to use the sulfonamide compounds in the treatment of sinus disease will depend on the comparable value of these highly efficient drugs as weighed in the balance against the menace of toxic reactions and the danger of creating a state of sensitivity in the patient.

It is well to review present knowledge of the action of the sulfonamide compounds from time to time in order that one's treatment may not become a ritual but rather a specific therapy against a thoroughly understood pathologic condition.

The sulfonamide compounds act to produce bacteriostasis. The growth of the organisms is inhibited. In this way these drugs prevent early overwhelming poisoning and provide a safe waiting period until the forces of immunity can be organized to bring about recovery through the phagocytic powers of the body cells. In some manner the sulfonamide compounds interfere with the metabolism of bacteria, but this action is diminished or abolished in the presence of pus or necrotic material. It is well to remember this fact when one is dealing with purulent and necrotic material in a nasal sinus. Bacteria which have been retarded in their growth by sulfonamide compounds may regain their disease-producing characteristics when the administration of the drug is stopped, which may be interpreted as indicating a weak effort on the part of the phagocytic factors of the body and as a sufficient reason to bolster such weak forces by the transfusion of whole blood, the injection of immune serum or by other means.

Before instituting the chemotherapeutic treatment of sinus disease, the surgical features of the disease must be estimated. At this point one will remember that the sulfonamide compounds are ineffective in the presence of pus or necrotic material. Physicians have learned that the sulfonamide compounds are of no value in the treatment of abscess pockets or of infection which is closed off by the barriers created by thrombosed vessels. Experience with abscess of the lung, surgical mastoiditis and chronic osteomyelitis bears out this observation. The rhinologist will apply such surgical measures as are required to open up diseased sinuses preliminary to the oral use of the sulfonamide compounds. If he has followed surgical measures, he will treat the cavities produced by surgical treatment locally with the sulfanilamide crystals, keeping in mind that the cavities should not be filled with this material but should be given rather a dusting of the outlining walls.

A plan to be followed in the treatment of nasal sinus disease, whether this is of an acute or a chronic nature, must of necessity be an elastic one. Many factors enter into one's decision. Surgical treatment of diseased sinuses is well understood by the rhinologist, along with its dangers, limitations and unsatisfactory results in some cases. Whatever type of treatment is chosen, there are preparatory steps that must be observed. One of the more important of these is the determination of the bacterial flora of the nose and sinuses of the individual patient.

Material for bacteriologic study is procured by the surgeon himself. If the material for study is not collected by one familiar with the anatomy of the nose, the collecting swab will be contaminated by staphylococci from the skin, and a misleading report of the culture will be returned. A small malleable wire applicator, cotton-tipped, is used to collect the material from the middle meatus or from other likely areas. The material is cultured on blood agar and incubated under both aerobic and anaerobic conditions. It is of prime importance that the rhinologist know the offending organism, and in most cases there will be one type of organism that is predominant toward which he can direct the therapy. This marked predominance of a single type of organism is always observed in acute infections of the

sinuses. If the infections are long standing, a great variety of bacteria will be recovered, but by a careful bacteriologic study the pathogenic organisms can be identified, and the remaining saphrophytes can be cared for by simple methods of cleanliness.

In the treatment of nasal sinus infections, when the use of a sulfonamide compound is indicated, sulfadiazine is the drug of choice. This decision is made because of the low degree of toxicity and the specific action of this member of the sulfonamide group for the disease organisms that are usually the cause of sinus infections.

A satisfactory working rule for the daily dosage of sulfadiazine is 1 grain (0.06 Gm.) per pound of body weight. This rule of dosage applies to children as well as adults. The dose stated is considered a full dose. A moderate dose is ½ grain (32 mg.) per pound of body weight, while a small dose is ¼ grain (16 mg.) per pound of body weight. The drug concentration is the same in the tissues as in the blood stream, and it is in regard to the most effective concentration of the drug that physicians still have more to learn. They do know that gonorrheal infections are controlled by what are considered small doses of the sulfonamide compounds with low levels in the blood. It may be that other bacteria will be found to succumb to smaller amounts of these drugs than are now thought necessary.

In cases of fulminating sinus infections or other cases of a desperate nature in which medication by mouth is not possible or the drug cannot be retained, one can inject the sodium salt of sulfadiazine intravenously. Adults in this group are given 2.5 Gm. or 10 cc. of a 25 per cent solution at eight hour intervals. By whatever route the drug is given, one holds to the principle that a large initial dose, usually 4 Gm. of sulfadiazine, should be used and the treatment continued on the schedule of 1 grain per pound of body weight per day, medication being given at four hour intervals.

Sulfadiazine manifests a lower degree of toxicity than the other sulfonamide compounds and toxic reactions are not expected during the first seven days of treatment. The more serious side effects of the drug are: agranulocytosis, hemolytic anemia and disturbance of the functions of the kidneys. Eight per cent of patients will show some degree of toxicity from sulfadiazine, and about one half of this 8 per cent will have some renal irritation. The suppression of urine, the hematuria and the formation of crystals may be prevented in many cases by keeping the urine alkaline or neutral. The intake of fluid must be maintained at a high level. Careful estimates of the output of urine are an effective means of determining the functioning of the kidneys. Should the excretion of urine fall below 1,000 cc. in twenty-four hours, a revision of the treatment may be indicated.

The time has arrived when rhinologists must put aside what they have been pleased to call the conservative treatment of sinus infections. Sinusitis is a surgical infection and calls for the same active treatment that is given surgical infections in other parts of the body. The conservative treatment of sinus infections has been timid in its approach and barren of results. It is for this reason that I make use of the sulfonamide compounds to supplement the surgical treatment of these infections. Surgical measures are not indicated in cases of acute infection unless it be the breaking over of the middle turbinate bone. In cases of chronic infection, with a purulent discharge, an infected lining membrane and a sinus filled with necrotic material, the surgical removal of the products of infection will permit the maximum action of the sulfanilamide crystals applied to the operative field and of the sulfadiazine administered by mouth. This plan of treatment has enabled me to obtain marked improvement in many long-continued and obstinate infections of the nasal sinuses, with some clinical cures.

COMMENT

The sulfonamide compounds are not effective as bacteriostatic agents when used on the unbroken surface of the skin or of the mucous membrane. It is for this reason that it seems illogical to use solutions or suspensions of these drugs for local application to the unbroken mucous membrane of the nose with the expectation that disease-producing organisms which are in the soft tissues will be affected or controlled. The oral or the intravenous administration of these new chemical agents is the only means by which they can be brought in contact with the disease-producing organisms that are lodged in the lining membranes of the nasal sinuses. They must be used early in the acute infection, before the infected area is closed off by thrombosed vessels.

Rhinologists must review their pathology, revise their surgery and integrate their knowledge of the new chemotherapeutic agents. With proper timing of indicated surgical intervention coordinated with systemic use of the new drugs together with the application of selected bacteriostatic substances to the field of surgical operation, sinus infections of many types can be brought quickly under control.

SUMMARY

The local use of sulfadiazine in the concentration of 2.5 per cent in aerosol was found to be without effect in controlling the progress of nasal sinus disease, either of the acute or of the chronic type.

Wetting agents were used as vehicles to carry the sulfonamide compounds in the belief that their detergent characteristics would aid in the penetration of the ostiums of the sinuses. This experiment was a complete failure.

An attempt at forcing the sulfonamide compounds into these cavities by the displacement method of creating a partial vacuum in the sinuses was without success since contrast mediums and other solutions were found only in small quantities and in a limited number of the sinuses so treated.

The only successful mode of chemotherapy for nasal sinus disease was found to be that of giving full doses of the sulfonamide compounds by mouth. This treatment must be carried on under observation in a hospital where bacteriologic studies can be made, the blood level controlled and the blood picture and renal functions frequently observed. With this treatment, a large number of sinus infections both of an acute and of a chronic nature can be brought under control.

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BRONCHIAL ADENOMA WITH METASTASIS

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Among the endobronchial growths the so-called bronchial adenoma has come to be recognized as a fairly definite clinical and pathologic entity. Because bronchoscopic examinations and biopsies are performed more frequently today than in the past in dealing with doubtful pulmonary conditions, the diagnosis of bronchial adenoma is being made with increasing frequency. Owing to the gaps in present knowledge of tumors of this type, especially regarding their ultimate outcome, final decision as to the treatment of choice still remains unsettled. If these neoplasms are potentially malignant, as some claim, the more certain method of eradication by lobectomy or pneumonectomy should be given serious consideration in preference to any of the forms of endoscopic treatment, such as bronchoscopic removal, electrocoagulation and implantation of radon seeds.

Many articles on this subject have appeared since Wessler and Rabin [1] in 1932 emphasized the benign characteristics of these tumors. Taking the clue from these authors, bronchoscopists attempted to eradicate adenoma endoscopically. Total extirpation, however, was seldom accomplished by this treatment, principally because the extrabronchial portions, frequently extensive, could not be reached.

Pathologic studies, as well as clinical experiences, led some investigators to the belief that, after all, bronchial adenoma was an essentially malignant, although slowly growing tumor. Even in recent literature conflicting claims are still undergoing discussion. In view of the unsettled opinions a case in which malignant features developed in bronchial adenoma, as confirmed at necropsy, should be of interest and is reported herein. The tumor was first discovered in the right bronchus of a 28 year old woman. It caused her death thirteen and a half years later, after having metastasized extensively to the left lung. The postmortem diagnosis was adenocarcinoma. The report of another case recently encountered is appended to illustrate the uncertainty of both clinical and pathologic recognition. The specimen obtained at biopsy was thought to be carcinoma, whereas more careful scrutiny of the tumor after pneumonectomy disclosed that it belonged in the category of bronchial adenoma.

From the University of Colorado School of Medicine and Hospitals.

1. Wessler, H., and Rabin, C.: Benign Tumors of the Bronchus, Am. J. M. Sc. **183**:164-180 (Feb.) 1932.

REPORT OF CASES

CASE 1.—Mrs. E. S., aged 40 years at the time of her death at the Colorado General Hospital, Denver, on Aug. 31, 1941, first presented herself at the Lenox Hill Hospital, New York, on Feb. 27, 1930, at the age of 28, on account of cough and pain in the right side of her chest of two years' duration. These symptoms commenced after an attack of right-sided pneumonia two years previously. There had been no other severe illness and no operation. The one child was living and well. The family history was recorded as irrelevant.

Bronchoscopy performed at the Lenox Hill Hospital on March 10, 1930 disclosed a tumor at the entrance to the right main bronchus. Report on the tissue removed at bronchoscopy showed "adenomatous hyperplasia of the mucous glands, possibly carcinoma." Bronchoscopic treatments consisting of forceps removal, electrocoagulation, and implantation of radon seeds were given from March 14, 1930 to May 27, 1937. On Sept. 28, 1936 a pathologic diagnosis of "adenoma (cylindroma) of the bronchus" was recorded. On Jan. 9, 1937 the final pathologic diagnosis was "adenoma of the bronchus" (fig. 1). Roentgen treatments were administered from June 8 to June 24, 1937 (twelve treatments of 200 roentgens each, six to the anterior and six to the posterior tracheal regions).

The patient then moved to Denver, and when she was first examined bronchoscopically at the National Jewish Hospital, April 15, 1938, the entrance to the right main bronchus was seen to be narrowed to about the size of a pinhead. This narrowing was apparently produced by the extension of the tumor and by the contraction of fibrous tissue resulting from the previous treatments.

At this time the patient complained of dyspnea and wheezing in addition to the cough and pain in the right side of her chest. Bronchoscopic dilation of the strictured right bronchus, repeated at intervals of four to six months, resulted in only temporary relief.

On April 3, 1941 her general condition was rather poor, with marked loss of weight, increasing dyspnea and wheezing.

On April 15, since her dyspnea and cyanosis had become aggravated in spite of administration of oxygen, she was removed to the Colorado General Hospital for

a series of treatments with high voltage roentgen rays. These treatments were given from April 17 to May 3, eight to the anterior and seven to the posterior thoracic area, with a total of 4,200 r.

The woman was readmitted to the Colorado General Hospital on August 14, having experienced only slight improvement as a result of the previous roentgen therapy. The attacks of dyspnea, cough and pain in the chest had recurred at frequent intervals. She was again placed in an oxygen tent, and further palliative roentgen irradiation was started, especially of the left lung field, because the increased density was observed to be much more marked than in previous roentgenograms, extending now in fanlike manner from the left hilar region. (Previously, roentgenograms of the chest had shown only clouding of the right lung field, with

Fig. 1 (case 1).—Biopsy specimen of so-called bronchial adenoma, five years before the death of the patient.

displacement of the heart and aorta to the right, due to atelectasis; fig. 2.)

Necropsy.—The patient died on Aug. 31, 1941. The pertinent observations at necropsy, performed that same day, follow:

(*a*) Pleural Cavities: The right pleural cavity was obliterated by moderately firm fibrous adhesions which united almost the entire right lung to the parietal pleura. The left cavity, free from adhesions, contained between 300 and 400 cc. of clear yellow fluid, shifting the lung toward the midline.

(*b*) Trachea and Bronchi: Both the trachea and the bronchi were approximately normal in width and had a pale, glistening mucosa. Changes in the right bronchus near the hilus will be described with the lungs.

(*c*) Lymph Nodes: Mediastinal and peribronchial lymph nodes showed some anthracosis but were not enlarged. They revealed no tumor tissues macroscopically.

(*d*) Lungs: The right lung weighed 240 Gm. and was bilobar. The pleura was thickened and dull over the entire lung, particularly over the posterior aspect of the lower lobe, where also there were a few subpleural petechiae. The right bronchus was extremely narrowed at the hilus; its wall was firm and thickened and appeared a homogeneous grayish white. The lumen was scarcely pervious to a probe of small caliber. The involved portion of the bronchus was short, only about 1 cm. in length. The intrapulmonic bronchi appeared normal. On section, yellowish gray, rather firm, nonaerated tissue extended fan-shaped from the hilus into the upper lobe, occupying about one half of it and forming a mass about 4 to 5 cm. in diameter (fig. 3). This tissue nowhere extended to the pleura. It was fairly well demarcated from the normal, light red, aerated tissue, which occupied the remainder of the lung. Intrapulmonic vessels appeared intact.

The left lung weighed 325 Gm. The pleura was slightly thickened and rather dull. The bronchi and vessels showed no pathologic changes. Yellowish gray, firm tissue spread from the hilus into both lobes, occupying a zone 7 by 5 by 4 cm. in dimensions. This tissue resembled that on the right but was softer and spotted with tiny yellow dots; it gradually disappeared into normal tissue as did that on the right. In the posterior portion of the lower lobe a rather firm round nodule, 1.5 cm. in diameter, was seen; it was well demarcated, homogeneous and gray-yellow; its lower border was located about 2 cm. superior to the base of the lung. It was apparently independent of the

bronchi. After fixation of the lungs, three small, yellow-gray, firm tumor nodules, about 0.8 cm. in diameter, were found in the upper lobe close to its anterior surface.

Microscopic Observations.—In the stenosed portion of the right bronchus the lining was fairly well preserved; it consisted in part of flat cuboidal epithelium but mainly of three to four layers of squamous epithelium. The tumorous infiltration (fig. 4) spared both lining and cartilage. Otherwise it was diffuse, exhibiting small distorted glands or small solid nests and cords. Most of the tumor cells were low cuboidal, with medium-sized round or oval nuclei. Much of the fibrous stroma was hyaline; some, however, was more cellular and somewhat myxomatous in the vicinity of the lumen, where solid nests of tumor cells prevailed. Mucoid or hyaline material was present in many of the glandular spaces of the neoplasm. Tumor cells were found in some of the lymphatics, particularly those of the adjacent anthracotic lymph nodes.

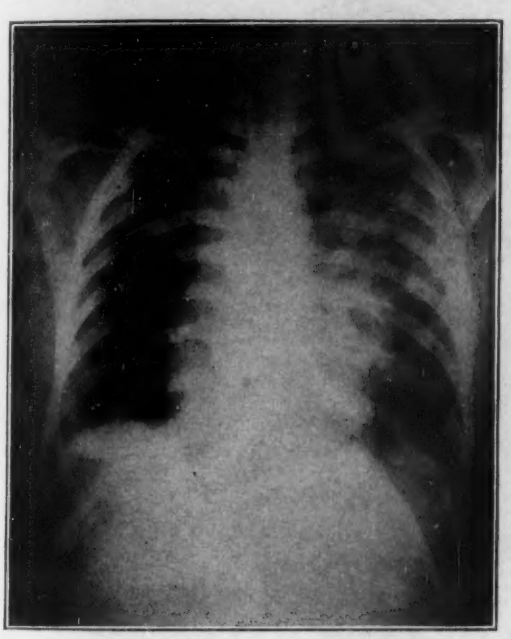

Fig. 2 (case 1).—Roentgenogram of the chest two weeks before the death of the patient, showing clouding of the right upper lung field, with displacement of the heart and aorta to the right, due to atelectasis. The increased density extending in fanlike manner from the left hilar region suggests metastatic involvement of the left lung.

The region of the lung close to the stenosed right bronchus was occupied by tumor tissue similar to that described in the foregoing paragraph. However, glandular structures were somewhat more prominent, the walls consisted of several epithelial layers, and the stroma was more cellular.

The nodular focus in the lower lobe of the left lung was made up of tumor tissue; there was less stroma than in that of the right lung, and the glandular lumens were larger and contained much mucoid and hyaline material. The focus in the left hilus contained no tumor tissue but showed irregularly thickened alveolar walls with much fibrous tissue; the alveoli were distorted in part and variable in diameter; they contained homogeneous masses or accumulations of phagocytes; many alveoli displayed a low cuboidal lining. Some spots in this area were entirely sclerosed and failed to reveal any recognizable alveolar structures.

Case 2.—Mrs. M. B., aged 45, entered the Colorado General Hospital, Denver, on Nov. 12, 1943 for pneumonectomy on the left side. A diagnosis of tumor of the left lung had been made elsewhere. The pulmonary trouble had commenced about one year previously, with cough, lassitude and loss of appetite. The patient had lost 40 pounds (18 Kg.) in the preceding year. Two months before admission to the hospital, on three different occasions she coughed up small amounts of blood. As a rule, the cough was nonproductive.

No previous serious illnesses were reported. Five children were living and well; there had been one miscarriage. The woman's husband died at the age of 56 years from cancer of the urinary bladder.

Bronchoscopic examinations by two bronchoscopists had been performed prior to admission to the Colorado General Hospital: the one reported that there was no evidence of bronchial tumor; the other observed a growth in the left main bronchus but was unable to obtain a specimen for biopsy.

Fig. 3 (case 1).—Probe in the narrowed bronchus; tumor tissue extending into upper lobe.

Artificial pneumothorax had been instituted on the left side ten days prior to admission. The left side of the chest was hyperresonant, there was decreased vocal fremitus, the movement of the diaphragm was restricted on that side and no vesicular breathing could be heard on the left side.

The physical examination otherwise was reported as yielding essentially negative results.

The urinalysis, the blood count and the serologic tests revealed no striking abnormalities.

Roentgenograms of the chest showed pneumothorax on the left side with practically complete collapse of the apex of the lung and marginal air visualization in the costophrenic angle and laterally. Increased radiopacity of the base of the left lung suggested tumor. The heart and the right lung appeared normal. There was no evidence of pleural effusion.

On November 19 one of us (H. I. L.) made a bronchoscopic examination and found a rounded smooth mass, the size of a small cherry, protruding from the orifice of the bronchus of the upper lobe of the left lung and obstructing about two thirds of the lumen of the bronchus of the lower lobe. Specimens removed at biopsy were reported as showing "probably oat cell carcinoma."

Pneumonectomy was performed on the left side on November 30 by Dr. George B. Packard. The patient has made an apparently good recovery.

The gross and microscopic observations on the lung removed at operation follow:

The specimen consisted of an entire left lung, measuring 14 by 13 by 3 cm. and weighing 270 Gm. The pleural surface was moderately thickened and slightly edematous. In the crotch at the first bifurcation of the main bronchus was a firm papillary pink mass, approximately 1 cm. in diameter, which projected into and partially obstructed the lumen. The surfaces on section showed apparent extension of this mass into the deeper bronchial and peribronchial tissues. In the upper lobe the smaller bronchi distal to the mass showed much dilatation and sacculation, measuring as much as 1 cm. in diameter, and were filled with thick gray mucoid material. The mucosal lining was slightly wrinkled and pale gray. The dilatations of the bronchi extended to the periphery of the lung. The blood vessels had thin and intact walls. In the lower lobe there was only mild dilatation of the smaller bronchi. Along these bronchi and in the parenchyma of the lung were irregular, pale yellow, cheeselike islands of tissue, 0.7 cm. or less in diameter. The whole lung was collapsed, the upper lobe more than the lower lobe, which was slightly crepitant. Surfaces on section were red-gray in the lower lobe and pale red-gray in the upper lobe. The base of the lower lobe was ragged, denuded of pleura and red-gray.

The papillary tumor was made up of anastomosing cords, rows and nests of cells (fig. 5). Numerous elongated, slender strands with a double band of cells were observed. Small papillae, each with a central vessel, were occasionally seen. The stroma was moderately well developed and consisted of straight or curved strands of poorly nucleated, often hyaline connective tissue. The tumor cells were polyhedral, ovoid or sometimes round. Although they tended to coalesce, in several fields isolated cells were discernible. The nuclei were regular, medium sized, generally round and fairly pale; they contained a slight to moderate amount of chromatin. Occasional nuclei were small and dark. A very small number of "palisade cells" were observed, with cone-shaped cytoplasm and elongated nuclei; the typical palisade effect, "with each alternate cell pointing in a direction opposite to its neighbor" (Stout[2]), however, was not observed. Giant cells and mitotic figures were absent. Neoplastic cells infiltrating through the bronchial wall were evident. The inner surface of the bronchus was in part composed of tumor tissue; at one point it was ulcerated and was covered by a moderately thick layer of fibrin, hematogenous cells and mucus. In this vicinity, small patches of the mucosa were still visible and exhibited squamous cell metaplasia. Other observations included peribronchial masses of foamy macrophages in various areas of the lung, secondary to an injection of iodized poppyseed oil.

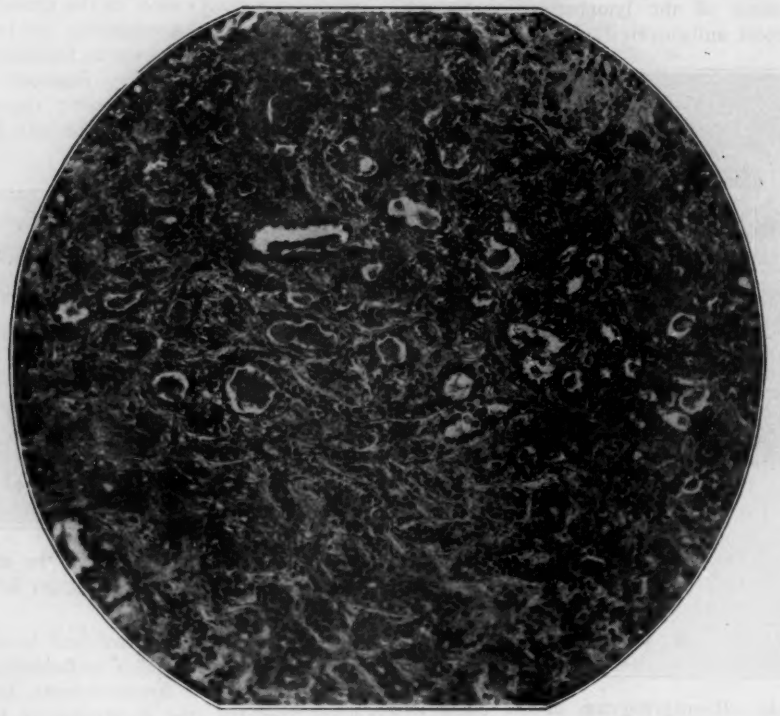

Fig. 4 (case 1).—Cylindromatous appearance of tumor.

COMMENT

Observations in 2 other cases in which necropsies were performed by one of us (K. T. N.) in former years were essentially similar. In both instances bronchial adenoma was incidentally discovered at necropsy. One of the tumors showed the "palisade cells" somewhat more numerous and the stroma more prominent and more vascular.

Our experience in cases 1 and 2 emphasizes the ease with which confusion as to the nature

2. Stout, A. P.: Cellular Origin of Bronchial Adenoma, Arch. Path. **35**:803-807 (June) 1943.

of bronchial adenoma can arise: the one which for many years had been regarded as benign, terminated with metastatic manifestations; the other, at first reported malignant, was ultimately found to be the so-called benign bronchial adenoma. Regarding the second case, it is obvious that more extensive study of the gross and microscopic aspects of the tumor following pneumonectomy necessitated a change in the diagnosis, revealing it to be one of the forms of bronchial adenoma rather than "oat cell carcinoma," as the biopsy purported to indicate.

It should be stressed that differentiation of benign from malignant tumors of the bronchi is not always possible from the bronchoscopic biopsy material alone. This material is likely to show distorted cells with loss of structural pattern and without clearcut relation to the adjoining normal tissue. Furthermore, it offers no criteria as to the amount of tumor invading the adjacent bronchial wall or as to the presence of local or remote metastases.

The ultimate outcome of the first case—in which an apparently "benign" bronchial adenoma terminated in metastasis and death—deserves more detailed consideration.

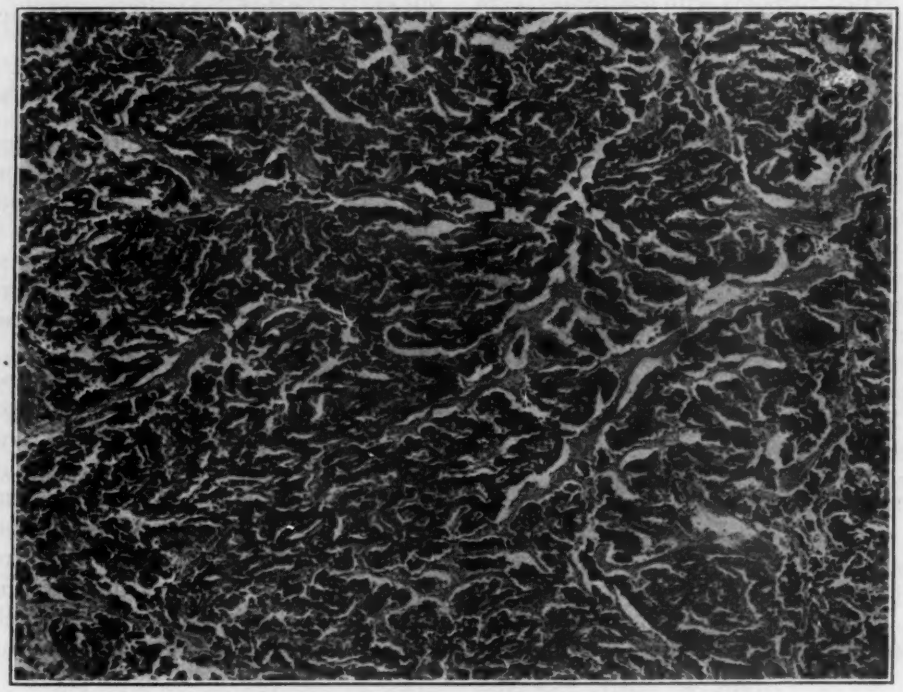

Fig. 5 (case 2).—Predominantly solid form of "bronchial adenoma."

REVIEW OF THE LITERATURE

After Mueller,[3] in 1882, described a pedunculated tumor observed in a bronchus at postmortem examination and diagnosed as adenoma, further reports remained scarce for nearly half a century. In 1930 Kramer [4] described the first cases in which such a tumor was diagnosed clinically and treated successfully, mainly by bronchoscopic removal. Larger series of cases were described by Geipel [5] in Germany in 1931, chiefly from the pathologic point of view. In this country Wessler and Rabin [1] in 1932 furnished the first comprehensive clinical picture and emphasized the benign characteristics of bronchial adenoma. Since then the literature has been extensive. In 1941 Foster-Carter [6] reviewed 87 cases and added 22 of his own. Between 1941 and 1943 many additional reports were recorded. In view of the fact that not all experiences with bronchial adenoma are published, its frequency must be fairly high.

A confusing nomenclature has arisen in connection with neoplasms of this type. They have been variously called: sarcoma of alveolar pattern, cylindric cell carcinoma, basal cell carcinoma, benign bronchial adenoma, carcinoid, adenocarcinoma, benign glandular tumor, vascular adenoma, endothelioma, mixed tumor, cylin-

3. Mueller, H.: Zur Entstehungsgeschichte der Bronchialerweiterungen, Inaug. Dissert., Ermsleben a. H., A. Busch, 1882.

4. Kramer, R.: Adenoma of Bronchus, Ann. Otol., Rhin. & Laryng. **39**:689-695 (Sept.) 1930.

5. Geipel, P.: Zur Kenntnis der gutartigen Bronchialtumoren, Frankfurt. Ztschr. f. Path. **42**:516-544, 1931.

6. Foster-Carter, A. F.: Bronchial Adenoma, Quart. J. Med. **10**:139-174 (July) 1941.

droma, reserve cell tumor and polypoid bronchial tumor.

Although the majority of writers now incline to the view that bronchial adenoma cannot be a strictly benign tumor, some still regard it as essentially such. Thus Foster-Carter [6] stated that bronchial adenoma may be regarded reasonably as a benign growth which does not cause death by direct or metastatic extension. Mason and Coberth [7] stated that they were not convinced that the rare reported cases of possible malignancy of this type of neoplasm have been adequately proved to have been that. Clerf and Bucher [8] concluded that bronchial adenoma is an epithelial tumor that is probably benign and nonmetastasizing. Brunn and Goldman [9] stated that cases of bronchial adenoma with metastases were probably not cases of adenoma originally.

DIAGNOSIS AND TREATMENT

Bronchial adenoma is commonly found in one of the main bronchi, is slow growing and produces severe cough, pain in the chest, wheezing dyspnea and frequently severe hemoptyses. Prolonged bronchial obstruction ultimately causes extensive damage to the lung distal to the stenosis, resulting in atelectasis, drowned lung, pneumonitis, bronchiectasis, pulmonary abscess and empyema.

The importance of early diagnosis has been stressed. Many authors have stated that cures can be effected by bronchoscopic removal in the early stages. Such a favorable outcome, however, has not generally been observed. Removal by forceps has been followed by brisk hemorrhage, and treatment by electrocoagulation has caused such severe secondary exsanguination, ten to twelve days later, as one of us [10] has reported elsewhere, that one hesitates to advocate this form of therapy seriously, especially since it is rarely totally curative.

Endoscopic treatment may result in cure when the neoplasm exists only as a pedunculated polyp. This rarely occurs; in most instances bronchial adenoma is sessile and has extrabronchial extensions, frequently larger than the endobronchial portions. The bronchoscopist, having no certain way of determining this important fact, can produce only a temporary reduction in the size of the intrabronchial portion of the tumor, and thus improve drainage and ventilation to the distal portion of the lung. Repeated use of forceps, electrocoagulation and implantation of radon seeds may cause an abundance of scarring and result in cicatricial contraction and bronchostenosis, as was demonstrated clearly in our first case. It appears that total eradication is dependent on intrathoracic surgical intervention. Many apparently permanent cures have been effected in these as well as in cases of bronchogenic carcinoma, which, of course, is not to be confused with bronchial adenoma. In contrast to the age incidence of the more common bronchogenic carcinoma, the adenoma endangers chiefly the female sex and the younger age groups, between 20 and 50 years.

PATHOGENESIS

Bronchial adenoma was thought from the first to be a tumor of the bronchial mucous glands and their ducts, although Jackson and Konzelmann [11] up to 1937 had not yet been convinced of such an origin. Because of a superficial resemblance to fetal lung tissue, Churchill [12] in 1937 thought that the tumor might represent a vestigial lobe in the bronchial wall.

Womack and Graham [13] called bronchial adenoma potentially malignant and chose to compare it to mixed tumor of the salivary glands on the basis of histologic observations, stating that it contained both entodermal and mesodermal structures. They showed specimens containing cartilage and bone, in addition to the grouping of the typical cuboidal or polygonal cells in varying patterns of alveolar, columnar, medullary, acinous, mosaic and angiomatoid arrangements. Clerf and Bucher [8] did not find bone and cartilage in any of their specimens. Stout [2] agreed with Mallory [14] that the bone and cartilage observed may result from metaplasia of the stroma.

Adams, Steiner and Bloch [15] called tumors of this type "malignant adenomas of the lung."

7. Mason, D. G., and Coberth, T.: Adenoma of the Bronchus with Successful Pneumonectomy: Report of a Case, Arch. Surg. **45**:542-550 (Oct.) 1942.

8. Clerf, L. H., and Bucher, C.: Adenoma (Mixed Tumor) of Bronchus: A Study of 35 Cases, Ann. Otol., Rhin. & Laryng. **51**:836-850 (Sept.) 1942.

9. Brunn, H., and Goldman, A.: Bronchial Adenoma, Am. J. Surg. **54**:179-192 (Oct.) 1941.

10. Laff, H. I.: Benign Tumors of Bronchi, with Special Reference to Vascular Adenoma, Arch. Otolaryng. **31**:148-159 (Jan.) 1940.

11. Jackson, C. L., and Konzelmann, F. W.: Bronchoscopic Aspects of Bronchial Tumors with Special Reference to Bronchial Adenomas, J. Thoracic Surg. **6**:312-329 (Feb.) 1937.

12. Churchill, E. D.: Discussion of Symposium on Surgery of the Lungs and Mediastinum, J. Thoracic Surg. **6**:335 (Feb.) 1937.

13. Womack, N., and Graham, E.: Mixed Tumors of the Lung, Arch. Path. **26**:165-206 (July) 1938.

14. Bronchial Adenoma, Cabot Case 27511, New England J. Med. **225**:983-986 (Dec. 11) 1941.

15. Adams, W. E.; Steiner, P. E., and Bloch, R. G.: Malignant Adenomas of Lung: Carcinoma-Like Tumors with Long Clinical Course, Surgery **11**:503-526 (April) 1942.

Among 5 cases, metastasis occurred in 3; in 1 to the bone marrow, in 1 to a tracheobronchial lymph node and in 1 to the lymph nodes adjacent to the lung and to the liver.

Mulligan and Harper [16] expressed the belief that bronchial adenoma originates from the bronchial glands; they called it slow-growing adenocarcinoma and mentioned its similarity to mixed tumor of the salivary gland.

Stout [2] recently contributed another point of view, reverting to the work of Hamperl,[17] who divided tumors of this type into two groups: those of cylindromatous form (analogous to the cylindromatous form of mixed tumor of the mucous and salivary glands), derived from the bronchial mucous glands, rarely encountered, and those of the usual form composed of solid cords and strands of tumor cells, tending to anastomose. Stout expressed the opinion that the growth may originate from the "onkocyte," a cell originally described by Hamperl. According to him, "onkocytes are epithelial cells which resemble those of the organ in which they are found but are larger and distinguished by having in their cytoplasm distinct, markedly acidophilic granules and nuclei which either look like the nuclei of the surrounding cells or which may be more deeply stained or even appear pyknotic." [2] Stout has been able to demonstrate onkocytes among the mucous and serous glands of adult human bronchi and their ducts and has expressed the belief that they may be the stem cell for the more common form of bronchial adenoma.

The sections in case 1 seem to indicate that the tumor is the rare form classed as cylindroma, because it certainly differs in histologic structure from those in the more common group, as shown in figure 5. Inasmuch as the two groups differ in the cellular structure of the tumors, it is reasonable to assume for them varying potentialities of tumor behavior. Perhaps the cylindromatous form only is capable of metastasizing, while the more common form tends to remain less malignant, as many authors contend.

At any rate, we wish to stress the potentially malignant character of neoplasms diagnosed as bronchial adenoma, manifested in their invasive growth (extrabronchial extension) and their ability to produce late metastases. Their histologically "benign" appearance is not incompatible with biologically malignant behavior (Rohdenburg [18]). A satisfactory name for these tumors has not been proposed as yet, and we are not inclined to contribute new suggestions to the already confusing terminology. The term "carcinoid" (first proposed by Hamperl) appears more justified than "adenoma," if used with the addition "bronchial" and with the understanding that one is dealing with a carcinoma-like tumor which may be biologically (but not histologically) similar to the carcinoid of the vermiform appendix.

SUMMARY

In the 2 cases of so-called bronchial adenoma reported the neoplasm presented different histologic patterns. In one the rare cylindromatous form existed for thirteen and a half years and terminated with metastasis to the opposite lung. In the second the more common solid form of the neoplasm was at first mistaken for "oat cell carcinoma."

The opinion of some investigators that bronchial adenoma is always a benign tumor is rejected.

Because of the potentially malignant characteristics, early lobectomy or pneumonectomy is considered the treatment of choice.

Correlated clinical and pathologic studies may serve to confirm the impression that the cylindromatous form is potentially more malignant than the more common form.

The term "bronchial carcinoid"—already employed by some authors—might be adopted in preference to "bronchial adenoma."

620 Metropolitan Bldg.

16. Mulligan, R. M., and Harper, F. R.: The Morphology of Primary Carcinoma of the Human Lung, J. Thoracic Surg. **12**:734-752 (Dec.) 1943.

17. Hamperl, H.: Ueber gutartige Bronchialtumoren (Cylindrome und Carcinoide), Virchows Arch. f. path. Anat. **300**:46-88, 1937.

18. Rohdenburg, G. L.: Personal communication to the authors.

EPIPHORA OF NASAL ORIGIN

A SIMPLE METHOD OF TREATMENT

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Frequently epiphora is a troublesome symptom from the physician's as well as the patient's point of view. Quite naturally a person suffering from tearing seeks the advice of an ophthalmologist, but in many cases it is also necessary to consult a rhinologist.

It is true that in the majority of cases the tearing results from local stenosis of the lacrimal duct, but not infrequently the cause is to be found in the nose. In these cases treatment with mild anesthetics or astringents or dilation by means of probes is not sufficient.

From the rhinologic standpoint, swelling or polypoidal thickening just under the anterior end of the inferior turbinate is liable to produce tearing. In other words, any mechanical obstruction at the nasal end of the lacrimal duct is of particular interest to the rhinologist.

To summarize the anatomy:

A normal nasolacrimal passageway presents the following:

(1) The two lacrimal canaliculi, running from the free margin of the upper and lower lid. These run at first vertically for a short distance, and then turn at an angle to run a medial course converging toward the lacrimal sac. They then unite either in a common trunk to empty into the sac, or they empty separately.

(2) The lacrimal sac is the upper dilated end of the nasolacrimal duct and is lodged in a deep groove formed by the lacrimal bone and frontal process of the maxilla. It is oval in form and measures about 12 mm. in length. Its upper end is closed and rounded, while the lower end continues as the nasolacrimal duct.

(3) The nasolacrimal duct extends from the lower part of the lacrimal sac to the inferior meatus of the nose, being slightly narrowed at its lower extremity.[1]

The inferior or nasal opening of the duct is variable in shape and position and is often difficult to find on inspection.

. . . In the foetus up to eight months, often up to birth, and sometimes even in the new born child, the duct is closed below by a thin partition formed by the approximation of the lining membrane of the nose to that of the duct, the ostium being subsequently formed by its dehiscence. The resulting opening, which is rarely as large as the lumen of the duct, may be round or linear, vertical or transverse, punctiform or even duplicate. Holmes found it nearly always slit-like and very fine; it may be guarded by a valve, a flap or a diaphragm.[2]

From the point of view of local treatment it is important to know the probable site of the nasal ostium.

Swerschewsky[3] described three positions: The first is in the roof of the inferior meatus of the nose, coinciding with the aperture of the bony nasolacrimal canal, and is found in 45 per cent. There is a fold guarding the opening in 27 per cent of these, a diaphragm in 12 per cent and a circular orifice in 6 per cent. The second is below this point on the side wall of the inferior meatus in 49 per cent, the generally described position. The aperture is a wide one in 18 per cent, a half-open canal in 16 per cent and a small groove in 15 per cent. The last is the condition considered by him to be most favorable to disease. The third is far in front or behind, the ostium appearing as a small canal in 3 per cent.

The position mentioned in most textbooks is at about the middle of the side wall of the inferior meatus, 30 mm., or 1¼ inches, behind the lateral margin of the anterior nares and opposite the junction of the first and second quarters of the attached border of the inferior turbinate, which overhangs it. In view of this it is surprising that the opening is not injured more frequently in irrigating the antrum through the inferior meatal route, with resultant epiphora.

The roentgenogram shown in figure 1 was taken after outlining the three turbinates and the duct with lead oxide. The metallic paste is opaque to the rays and shows the length, the direction and the caliber of the tube, as well as the lower outlet in the nose.

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1. Campbell, D. M.; Carter, J. M., and Doub, H. P.: A Method for Roentgen-Ray Demonstration of the Nasolacrimal Passageways, Am. J. Roentgenol. 9:381-387 (June) 1922.

2. Whitnall, S. E.: Anatomy of the Human Orbit, London, Oxford University Press, p. 234.

3. Swerschewsky: Die anatomischen und pathologischen Verhaltnisse der Tranenableitungswege, abstracted, Jahresb. f. Ophth., 1910, p. 37.

In this connection a method of shrinking the inferior turbinate in cases of chronic obstructive rhinitis described by Thacker [4] is of particular interest to the rhinologist. He used fatty acid derivatives (sodium morrhuate, sylnasol [a 5 per cent solution of the sodium salts of certain of the fatty acids of the oil extracted from a seed of the psyllium group, to which 2 per cent benzyl alcohol is added for its anesthetic effect]

Fig. 1.—The lateral wall of the nose showing the turbinates and the position of the nasolacrimal duct. As shown by the roentgenogram, the duct extends downward and slightly backward from the anterior attached margin of the middle turbinate to open below in the inferior meatus.

and monoethanolamine oleate) as sclerosing solutions and reported excellent clinical results. The cases selected were those of the nonspecific and nonallergic type of rhinitis, the only indication being that the inferior turbinate showed definite shrinking after the use of astringents.

For the past two years I have used this method when the stenosis appeared to be at the nasal opening of the nasolacrimal duct. In uncomplicated cases, that is without polypoidal thickening of the inferior turbinate, good results are obtained. At times, if the inferior turbinate is crowded against the lateral wall, it is necessary first to fracture it to the midline, but in most cases the simple injection is sufficient.

According to Holmes,[5] who measured the position of the nasal opening of the duct in 50 specimens, the average was 6 mm. behind the anterior attached margin of the inferior turbinate and 16 mm. above the floor of the nose.

The technic is essentially the same as that described by Thacker.[4] After covering the anterior end of the inferior turbinate and the meatus with tampons saturated with an anesthetic solution, a no. 22 needle, about 2 inches (5 cm.) in length, is attached to a tuberculin syringe. (A bayonet-shaped needle is useful, as vision then is not impaired.)

A little of the sclerosing agent, usually 4 to 6 minims (0.25 to 0.37 cc.), is drawn into the syringe. After removal of the tampons the needle is inserted into the lateral wall of the inferior meatus so that the point pierces the mucous membrane about 5 or 6 mm. behind the anterior attached margin. The bevel of the syringe is turned away from the bony wall of the nose and the injection made.

Within a few minutes pain is felt at the site of injection and in the upper teeth on the same side. In a recent publication by McAuliffe, Goodell and Wolff [6] on experimental production of pain in the nose and sinuses a number of interesting points were elicited.

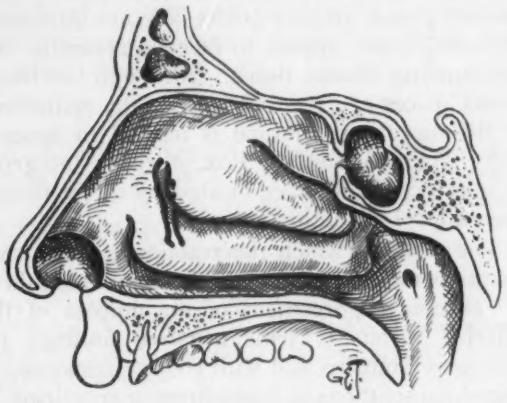

Fig. 2.—Diagram to show the position of the nasal opening of the lacrimal duct on the lateral wall of the inferior meatus as if seen through the overhanging inferior turbinate. In most cases, it is about 6 mm. behind the beginning of the anterior attached margin of the inferior turbinate and 16 mm. above the floor of the nose.

. . . The lower, the middle and the upper turbinates, whether stimulated mechanically with a probe, or by a faradic current, were considerably more sensitive than the nasal floor or the septum. A sharp burning pain was felt at the site of stimulation and along the lateral wall of the inside of the nose. A duller, aching pain was referred into the upper teeth when faradic current

4. Thacker, E. A.: Value of Fatty Acid Derivatives in Treatment of Chronic Obstructive Rhinitis, Arch. Otolaryng. **36**:336 (Sept.) 1942.

5. Holmes, C. R.: Extirpation of the Lacrimal Gland in Epiphora, Arch. Ophth. **48**:333 (July) 1919.

6. McAuliffe, G. W.; Goodell, H., and Wolff, H. G.: Experimental Studies on Headache: Pain from the Nasal and Paranasal Structures, A. Research Nerv. & Ment. Dis., Proc. **23**:185, 1943.

or pressure was applied to the anterior portion of the lower turbinate. When the middle and posterior portion of the lower turbinate were stimulated, pain also was felt under the eye, along the zygoma, and toward the ear.

After the injection of a small amount of the fatty acid solution the patient experiences profuse lacrimation with sometimes dilatation of the pupil and photophobia. This condition, which is due to ballooning of the tissue in the neighborhood of the duct, soon passes off. Within a few days shrinkage occurs, which tends to dilate the opening and, as a rule, improves the breathing on that side. This shrinking continues for five or six weeks and then remains stationary.

One or two points should be mentioned: In selecting a local anesthetic it is important to use one that does not shrink the mucous membrane. It is then much easier to inject the sclerosing agent without perforating the membrane, and the solution infiltrates the submucosal tissue much more readily. A 2 per cent solution of tetracaine hydrochloride or a 2 per cent solution of butacaine sulfate appears equally satisfactory. After shrinkage occurs, the tissue adjacent to the nasolacrimal duct microscopically shows increased fibrosis throughout the tunica propria. The mucous glands and the goblet cells are numerous but smaller and appear to be compressed by the surrounding fibrous tissue. Although the blood vessel spaces are reduced in size, the epithelium of the mucous membrane is normal in appearance. As mentioned by Fox,[7] there is "no gross or microscopic evidence of atrophy of the tissues even after 18 months."

The cases in which the reaction to sclerosing agents is most favorable are those in which there is "chronic engorgement of the mucosa of the inferior turbinate, with negative findings for allergy or sinusitis and with routine responses to shrinking solutions of epinephrine or ephedrine."[8]

At times it is necessary to treat a person more than once to obtain the desired result. If after two or three weeks the patient still complains of epiphora, it is advisable to inject another 4 to 6 minims of the solution under the anterior end of the inferior turbinate. As long as small amounts are used, no ulceration or sensitivity is likely to occur even on repeated injections.

The sclerosing agents which have been described are the ones used in the obliterating treatment of varicose veins—sodium morrhuate, sylnasol and monoethanolamine oleate (a 5 per cent solution with benzyl alcohol). In shrinking action the three solutions appear to be equally good. However, there are certain disadvantages to sodium morrhuate. It has a strong fishy odor, and if a little of the fluid runs into the pharynx, it is quite disagreeable and to some patients nauseating. Sylnasol is more suitable, as the taste and smell are not too unpleasant, and there are no allergic reactions from its use. Monoethanolamine oleate, which goes under the name of monolate, is very satisfactory, as it is uniform in consistency. This cannot always be said of sodium morrhuate, which is derived from cod liver oil and varies in composition, depending on the source of supply. Monolate has a strong sweetish odor resembling that of vanilla, but if any of the solution drops into the pharynx, the patient experiences an acrid taste and a burning sensation for a few minutes—similar to that when sylnasol is used.

This method of injection has given excellent results in the past two years without any harmful effects. It is strongly recommended in cases of tearing if the lacrimal duct seems patent after syringing or probing. For patients whose epiphora is apparently due to a localized narrowing at the nasal opening of the nasolacrimal duct, this form of treatment certainly should be considered. If polypoidal thickening of the inferior turbinate has taken place, and no reaction to shrinking agents is obtainable, better results are produced by means of submucosal electrocoagulation after infraction of the turbinate to the midline.

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7. Fox, S. L.: Chronic Vasomotor Rhinitis: A Clinical Investigation of Its Treatment with a Sclerosing Agent, Laryngoscope **53**:759 (Dec.) 1943.

8. Tamari, M., and Busby, W.: Evaluation of Submucosal Shrinking Methods for Chronic Nasal Obstructions, Arch. Otolaryng. **39**:331 (April) 1944.

THROMBOPHLEBITIS OF A CAVERNOUS SINUS FOLLOWING EXTRACTION OF TEETH

CURE WITH PENICILLIN AND HEPARIN

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OAKLAND, CALIF.

Prior to the day of sulfonamide compounds in therapy, recovery from thrombophlebitis of a cavernous sinus was extremely rare.[1] With the advent of these compounds reports of cures began to appear following the administration of sulfanilamide,[2] sulfapyridine,[3] sulfathiazole [4] and presumably sulfadiazine.[5] The use of anticoagulants, especially heparin, as adjuvant therapy seemed to enhance the value of the sulfonamide compounds,[6] but this was by no means proved.[4f] Recovery from cavernous sinus thrombophlebitis with penicillin therapy has been reported.[7]

The case reported now is noteworthy because (1) it illustrates again the danger of performing an operation on the maxilla and the palate in the presence of acute sinusitis, (2) thrombophlebitis of a cavernous sinus developed in the presence of an adequate sulfadiazine level in the blood and (3) a dramatic recovery followed the administration of penicillin and heparin. The paucity of reports of cases in which a patient was treated with penicillin for this condition justifies report of even a single instance of such successful treatment.

REPORT OF A CASE

W. F., an 18 year old Caucasian youth, was first seen in our clinic Jan. 3, 1944, with bilateral maxillary sinusitis. On January 5, antral irrigations revealed the presence of pus in both maxillary sinuses. Satisfactory progress was made until March 27 when an acute exacerbation supervened, with complaints of headache, backache and pain in the right upper teeth. The temperature was 100.2 F. The leukocyte count was 16,800 per cubic millimeter, with 69 per cent neutrophils. Conservative therapy was advised. However, on the next day the patient consulted his dentist, who

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The penicillin was provided by the Office of Scientific Research and Development from supplies assigned by the Committee on Medical Research for clinical investigations recommended by the Committee on Chemotherapeutic and Other Agents of the National Research Council.

1. MacNeal, W. J.; Frisbee, F. C., and Blevins, A.: Thrombophlebitis of the Cavernous Sinus: A Review of Reported Recoveries with Special Reference to Thrombophlebitis of Staphylococcic Origin, Arch. Ophth. **29**:231 (Feb.) 1943.

2. Seydell, E. M.: Recovery from Thrombosis of the Cavernous Sinus, Arch. Otolaryng. **30**:429 (Sept.) 1939. Barnshaw, H. P.: Report of a Case of Bilateral Cavernous Sinus Thrombosis: Recovery without Operative Intervention, J. M. Soc. New Jersey **36**:22 (Jan.) 1939. Pace, E.: Thrombosis of the Cavernous Sinus, Arch. Otolaryng. **33**:216 (Feb.) 1941.

3. Morrison, L. F., and Schindler, M.: Cavernous Sinus Thrombosis: Report of Recovery Following Sulfapyridine Therapy, Arch. Otolaryng. **31**:948 (June) 1940.

4. (*a*) Lyons, C.: The Treatment of Staphylococcal Cavernous Sinus Thrombophlebitis with Heparin and Chemotherapy, Ann. Surg. **113**:113 (Jan.) 1941. (*b*) Schall, L. A.: Treatment of Septic Thrombophlebitis of the Cavernous Sinus, J. A. M. A. **117**:581 (Aug. 23) 1941. (*c*) Wolfe, C. T., and Wolfe, W. C.: Thrombosis of the Cavernous Sinus with Recovery, Arch. Otolaryng. **33**:81 (Jan.) 1941. (*d*) Erschler, I. L., and Blaisdell, I. H.: Massive Hematuria Following Use of Heparin in Cavernous Sinus Thrombosis, J. A. M. A. **117**:927 (Sept. 13) 1941. (*e*) Skemp, H. I.; Afremow, M. L., and Rhoads, P. S.: Thrombosis of the Cavernous Sinus with Staphylococcic Septicemia Treated by Intravenous Injection of Sodium Sulfathiazole, with Recovery, Arch. Otolaryng. **34**:1025 (Nov.) 1941. (*f*) Moore, G. B., Jr.; Gardner, J. D.; Bell, K. R., and Tannenbaum, A. J.: Cavernous Sinus Thrombophlebitis: Cure with Sulfathiazole, J. A. M. A. **119**: 708 (June 27) 1942. (*g*) Weber, J. J., and Clinco, A. A.: Thrombosis of the Cavernous Sinus in the Presence of Pituitary Adenoma: Report of a Case with Recovery, Arch. Otolaryng. **36**:562 (Oct.) 1942. (*h*) Doorly, A. R. C.: Thrombophlebitis of the Cavernous Sinus: Recovery, Brit. M. J. **1**:42 (Jan. 9) 1943. (*i*) Cohen, M., and Cohen, H. J.: Septic Thrombophlebitis of the Cavernous Sinuses in a Two Year Old Child, with Recovery, Am. J. Dis. Child. **63**:546 (March) 1943.

5. (*a*) Heath, M.: Chemotherapy in Ophthalmology, J. A. M. A. **124**:152 (Jan. 15) 1944. (*b*) Zucker, H. D.: Clinical Experiences with Dicoumarol, ibid. **124**: 217 (Jan. 22) 1944. Schall.[4b]

6. Lyons.[4a] Schall.[4b] Erschler and Blaisdell.[4d] Cohen and Cohen.[4i] Zucker.[5b]

7. Florey, M. E., and Florey, H. W.: General and Local Administration of Penicillin, Lancet **1**:387 (March 27) 1943. Goodhill, V.: Penicillin Treatment of Cavernous Sinus Thrombosis, J. A. M. A. **125**:28 (May 6) 1944.

extracted the right upper first bicuspid tooth as well as a supernumerary tooth which lay alongside the bicuspid and which was impacted in the hard palate. The extraction was performed with the operative field under local anesthesia, apparently with some difficulty, as it occupied more than one hour.

Fig. 1.—Appearance of patient with thrombophlebitis of a cavernous sinus on the ninth hospital day.

The patient was next seen by us on March 30 (two days after the operation), at which time he appeared acutely ill. The temperature was 102 F., and cellulitis of the right cheek was apparent. The tooth socket was tightly sutured and grossly infected. To facilitate drainage the sutures were removed. Sulfadiazine therapy was begun, and massive local hot compresses were recommended. Hospitalization was refused by the patient until April 1.

Physical Examination.—On the day of admission the patient was acutely ill and in obvious distress. The temperature was 100.8 F. The right cheek was swollen, hot, tender and red. There was tenderness in the right infraorbital region. Thick yellow pus was draining from the right first bicuspid socket. The rest of the physical findings were not remarkable.

Laboratory Observations.—Cultures of the pus from the draining tooth socket were positive for Staphylococcus aureus. The leukocyte count was 14,800 per cubic millimeter, with 77 per cent neutrophils. The hemoglobin content was 102 per cent. The urine

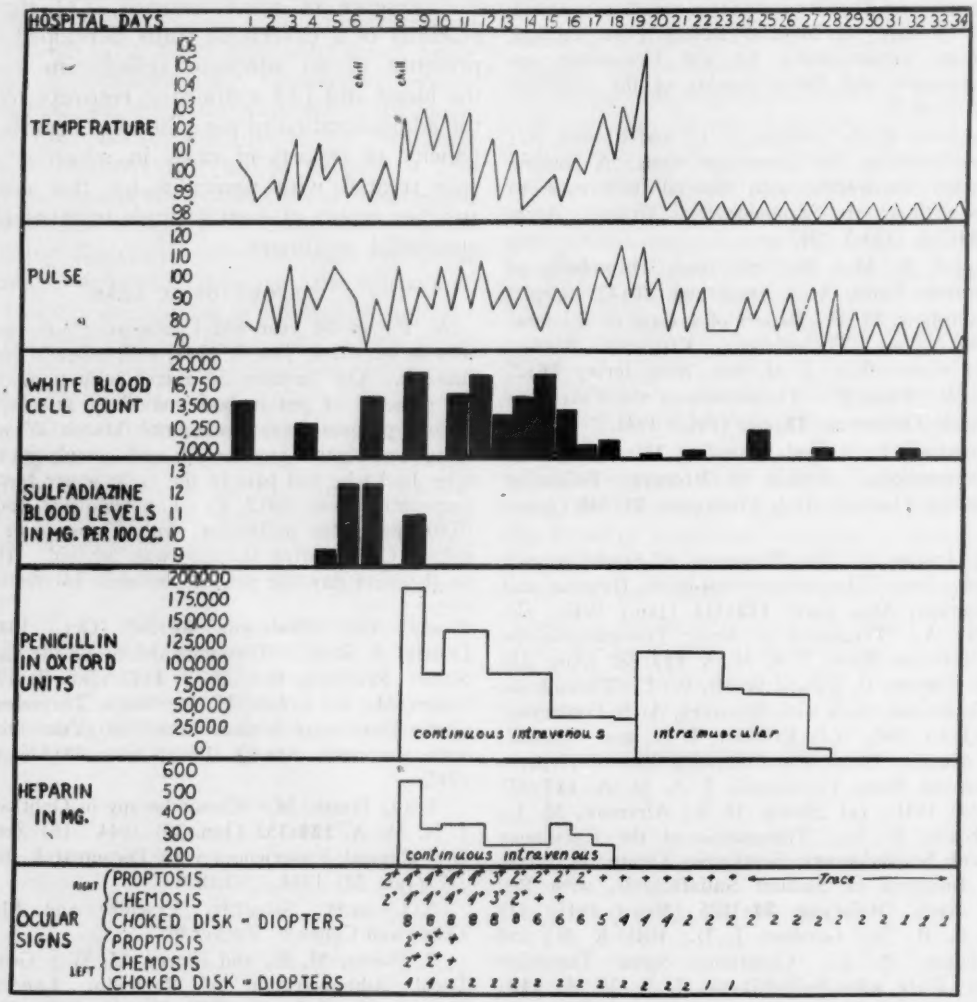
HOSPITAL DAYS
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34
TEMPERATURE
106 105 104 103 102 101 100 99 98
chill
chill
PULSE
120 110 100 90 80 70
WHITE BLOOD CELL COUNT
20,000 16,750 13,500 10,250 7,000
SULFADIAZINE BLOOD LEVELS IN MG. PER 100 CC.
13 12 11 10 9
PENICILLIN IN OXFORD UNITS
200,000 175,000 150,000 125,000 100,000 75,000 50,000 25,000 0
continuous intravenous
intramuscular
HEPARIN IN MG.
600 500 400 300 200
continuous intravenous
OCULAR SIGNS
RIGHT
PROPTOSIS
CHEMOSIS
CHOKED DISK - DIOPTERS
LEFT
PROPTOSIS
CHEMOSIS
CHOKED DISK - DIOPTERS
Trace

Fig. 2.—Clinical record with the schedule of penicillin and heparin therapy in a case of thrombophlebitis of a cavernous sinus. The ocular signs are graded by means of pluses: 4 plus means severe; 3 plus, moderately severe; 2 plus, moderate; 1 plus, mild.

showed a trace of albumin and 4 to 5 red blood cells per high power field.

Diagnosis on Admission.—The diagnosis was post-extraction infection of the right maxilla and adjacent soft tissues.

Course.—The patient was given large doses of sulfadiazine, with the blood level of the drug varying between 9.6 and 12.8 mg. per hundred cubic centimeters. Adjuvant therapy consisted of intensive nasal shrinkage, continuous local application of massive hot compresses, oral irrigations with hot isotonic solution of sodium chloride and administration of analgesics and sedatives. In spite of this, the infection spread and the patient continued to haye a septic type of fever. On April 6 he had a chill. The facial swelling extended into the right infraorbital area, and pus was now present in the right middle meatus. Proptosis of the right eye and chemosis of the bulbar conjunctiva became evident on April 8. The ocular fundi appeared normal. On April 9 the diagnosis of thrombophlebitis of the right cavernous sinus was established. On this day palsy of all the right extraocular muscles was noted. The proptosis and the chemosis increased. The right optic disk was found choked to 8 diopters, and the retinal veins were markedly distended. Several hours later edema of the left eyelids, and proptosis and chemosis of the left eye developed. The left optic disk was choked to 4 diopters. There were no signs of meningeal irritation. The patient lapsed into coma and appeared moribund.

A continuous intravenous drip of penicillin and heparin was started and continued for eleven days; 200,000 units of penicillin was given during the first twenty-four hours, 100,000 units during the next twenty-four hours, 150,000 units daily for the following two days, 100,000 units daily on the fifth, sixth and seventh days and 50,000 units daily for the four succeeding days. Further administration of penicillin was by intramuscular injection; 15,000 units in 3 cc. of isotonic solution of sodium chloride was given every three hours for the next four days. The dose was then reduced to 10,000 units, given every three hours for five days. A total dose of 2,000,000 units of penicillin was given in twenty days.

The dosage of heparin was adjusted to maintain the clotting time at approximately fifteen minutes; 600 mg. of heparin was given in the first twenty-four hours; 300 to 400 mg. was found to be adequate thereafter.

Pertinent clinical, laboratory and therapeutic data are graphically portrayed in figure 2.

After twenty-four hours of penicillin-heparin therapy the periorbital edema and chemosis on the left side were definitely subsiding, and the patient became more lucid mentally. After the seventh day the temperature began to rise in spite of the clinical improvement and a normal leukocyte count. We were unable to account for this except by the presence of thrombophlebitis of the antecubital vein which was the site of the intravenous infusion of penicillin. Perhaps the febrile episode was due to the continued intravenous administration of penicillin [8] or to the use of heparin.[9] The temperature fell to normal after the use of heparin and the intravenous injection of penicillin were discontinued. The administration of penicillin, as previously noted, was changed to the intramuscular route. After the twelfth day of penicillin therapy the patient became and remained afebrile. The facial swelling had almost disappeared by the thirtieth hospital day. The patient was discharged as cured on the thirty-fourth hospital day.

A blood culture on April 8 was positive but was contaminated before the bacterium could be identified. Subsequent blood cultures taken after the onset of penicillin therapy were all negative.

Roentgenograms taken on the twenty-fifth and thirty-first hospital days failed to reveal osteomyelitis of the right maxilla. However, a fragment of a root was demonstrated in the right upper first bicuspid tooth socket.

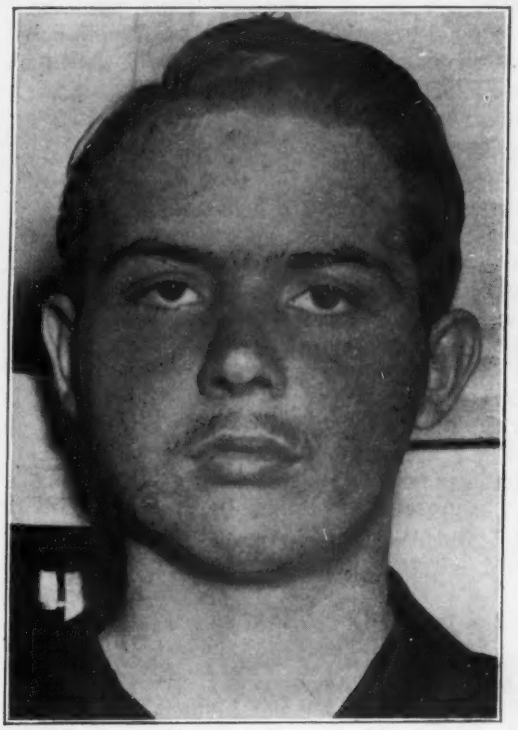

Fig. 3.—Appearance of patient on discharge from the hospital.

The proptosis of the right eye and the edema of the right side of the face had cleared completely three weeks after the patient left the hospital. There was no permanent disturbance of vision in either eye. When last seen, four weeks after discharge from the hospital, the patient was in good health and without complaints. A slight ptosis of the upper lid of the right eye, apparent in figure 3, and some discoloration of the right cheek were the only sequelae.

COMMENT

This case is illustrative of the well known dangers of operations on oral structures in the presence of acute infection.

8. (*a*) Keefer, C. S.; Blake, F. G.; Marshall, E. K., Jr.; Lockwood, J. S., and Wood, W. B.: Penicillin in the Treatment of Infections: Statement by the Committee on Chemotherapeutic and Other Agents, Division of Medical Sciences, National Research Council, J. A. M. A. **122**:1217 (Aug. 28) 1943. (*b*) Ericksen, O. C.: Staphylococcus Albus Osteomyelitis and Septicemia Treated with Penicillin, ibid. **124**:1053 (April 8) 1944.

9. Loewe, L.; Rosenblatt, P.; Greene, H. J., and Russell, M.: Combined Penicillin and Heparin Therapy of Subacute Bacterial Endocarditis, J. A. M. A. **124**: 144 (Jan. 15) 1944.

It is now well established that staphylococcic infections may be refractory to treatment with sulfonamide compounds. The organism may be primarily resistant or may acquire resistance to these drugs. It is in such cases that penicillin has its greatest value.[5a]

It is presumed that recovery of this patient was due to penicillin.

The role of heparin in the recovery is difficult to evaluate. The use of heparin as adjuvant therapy was instituted on the basis of the studies by Lyons [4a] and Schall,[4b] who thought that it was of definite value in the treatment of cavernous sinus thrombophlebitis.

No toxic reactions to penicillin-heparin therapy were observed other than mild thrombophlebitis of the antecubital vein and possibly the febrile episode, as previously noted.

SUMMARY

In a case of thrombophlebitis of the right cavernous sinus which followed extraction of teeth and which developed under sulfadiazine therapy, recovery followed treatment with penicillin and heparin.

Case Reports

CHRONIC EXTRADURAL ABSCESS IN CHILDREN

Its Effect on Behavior

JOHN B. PRICE, M.D., NORRISTOWN, PA.

During the course of the cases to be reported, it seemed striking to me that a number of children who suffered from chronic otitis media were hard to manage and were labeled as inferior. It became more striking when the otitis was surgically corrected and an extradural infection was discovered and treated; for the children's behavior showed marked improvement.

There is little relating to this subject to be found in the literature. In fact, it seems to be taken for granted that toxic processes in any part of the body will produce lethargy and dulness and to a considerable extent backward mindedness. It also seems to be the prevailing opinion that this is a field for the psychiatrist, who will unearth some hidden phobia to explain the behavior of these children. Yet it is possible that intracranial pressure and paracerebral infection produce a lethargic, moronic child who cannot seem to associate himself with other children. Such a child cannot make progress in school, and its behavior toward its schoolmates or other young associates is either indifferent or bad. As things are, the general rule is to find some institution or school for the child showing the deficiencies described.

Some years ago I was engaged in doing surgical work on the ear, nose and throat at the Pennhurst State School of Pennsylvania. This institution is concerned entirely with the training and treatment of substandard and mentally deficient children. Aside from their constitutionally inferior background, most of the children had sinus infection, bad teeth and infected tonsils, and a large number had chronically discharging ears. Mastoidectomy frequently revealed infection extending into the middle or the posterior fossa. Seven cases are presented here. Before I was aware of the importance of the implications in these cases, I unwittingly performed mastoidectomy on many without realizing what had happened to these boys and girls. Most of these children were boys between 12 and 20 years of age.

When one stops for a moment and thinks of the effect on young boys socially, economically and legally, one wonders whether a great many have not been unjustly condemned because of some paracerebral pressure.

My thesis is this: All infections surrounding the brain should be searched for and all chronically discharging ears should be explored and treated when these conditions are present in mentally backward children.

The first patient of this type I ever encountered was seen by me twenty years ago. The boy was a dull, witless youngster who sat all day playing with a string or some simple toy. His grandmother told me he had constant swellings about the ear as well as a discharge from the ears and the nose. With the help of a more experienced otologist, I operated on the right mastoid process. The inner table was necrotic and the dura of the middle fossa exposed. All necrotic bone was removed. The boy made a remarkable recovery. Two years later he became a leader among the children in his neighborhood, a fighting, active boy. Since then I have seen this happen frequently.

It is probable that the abnormal behaviorisms of such children are the result of an extradural abscess causing pressure on the brain. It is also peculiar that when these manifestations occur in children who are left handed the infection is on the right side and when they occur in right-handed children it is on the left side. The children are dull and have behaviorisms similar to those observed following injury of the brain. It is my belief that in these cases the ears should be dealt with surgically as soon as possible in order to prevent mental complications.

The following cases are all authentic. The records are taken from the files of the Montgomery Hospital.

REPORT OF CASES

CASE 1.—B. B., a girl aged 7, was operated on May 22, 1930. Since the age of 2 she had had chronically discharging ears. She was irritable, hard to manage, spent a great deal of time sulking and was extremely moody. Her ears had been treated intermittently without benefit. Bilateral mastoidectomy was performed by me, and necrotic bone with an extradural abscess was found on each side. The child made an excellent recovery. Her mentality improved, and at the date of writing, fourteen years later, she is a remarkable artist, has a fine and dignified approach and has to her credit many admirable paintings and charcoal sketchings.

CASE 2.—B. B., a boy, had a discharging ear for five years. He was listless and dull and was being considered a good subject for a school of mentally backward children. Simple mastoidectomy was performed on Nov. 1, 1935, and an extradural abscess was found after removal of some necrotic bone in the region of the middle fossa. Shortly after the operation the boy's attitude toward school and his companions assumed an entirely different aspect. At the date of writing, March 1944, his parents state that he

is a bright active young man and that he has been accepted as a cadet in the Naval Air Corps.

CASE 3.—H. F., a young boy, had had bilaterally discharging ears for four years. He was so unruly in school that the authorities refused to let him come to school. He constantly fought with his brothers, and no one could control him. On Jan. 18, 1942, bilateral mastoidectomy was done, and an extradural abscess was found on each side. Since the operation the boy has been readmitted to school, and while he is not a brilliant student, his habits and behavior have entirely changed.

CASE 4.—E. C., a boy aged 5, had acute otitis on the right side, which was permitted to become chronic. He became dull and apathetic and was absent from school most of the time. When an acute febrile exacerbation of the middle ear infection occurred, simple mastoidectomy was done, and a large necrotic area of bone was removed in the region of the middle fossa. At the present time the boy is doing grade A work in school.

CASE 5.—J. O., a boy aged 3, had had a discharging ear since the age of 18 months. He was listless and made no effort to understand or take an interest in anything. At the age of 3 simple mastoidectomy was done, and a large collection of pus was discovered extradurally. The child made a complete recovery and is now attending school as a normal boy.

CASE 6.—A. B., a boy aged 12, was listless, played aimlessly with objects and took no interest in any sort of conversation. He was absent from school more than half of the time. He had a discharging ear for three years. Mastoidectomy was done on June 18, 1939, revealing an extradural abscess. The boy is now a bright and intelligent young man.

CASE 7.—P. C., a boy aged 13, had a chronically discharging ear, was irritable and inattentive and got along with his family with difficulty. Mastoidectomy was done on the right side, and an extradural abscess was discovered. His behavior soon improved, and at the time of writing, six years after operation, he is apparently a normal young man.

This is but a small group of cases, but there must be many more which could be brought to attention once the mental behavior in some of the children suffering from chronically discharging ears is thought of in the light of the physical defect.

When this problem was discussed with Judge George C. Corson, of the Common Pleas Court, from the legal aspect, he agreed that a great deal of trouble and expense to the state could be obviated if a number of delinquent and criminal children could be improved by the correction of such physical defects.

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Clinical Notes; New Instruments and Technics

INSTRUMENT FOR ANCHORING FRACTURES OF BONES OF THE HEAD

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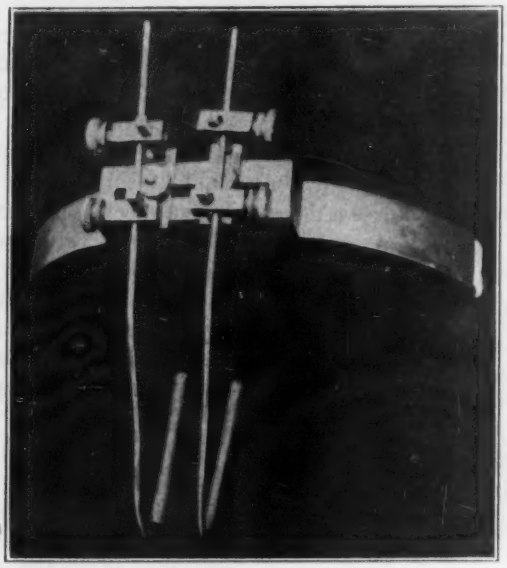
The problem of proper approximation and demobilization of bone fragments in certain cases of fracture of the mandible, the zygoma, the maxilla or other bones of the face has created a demand for an adjustable support in these cases. This support is best applied by an adjustable anchoring instrument which permits moving of the splint or of the traction wires in any direction so that the bone fragments can be brought into proper position. It holds the fragments stationary in the chosen position. The refined instrument which is now presented has been evolved through different stages in an effort to improve the results obtained in fractures of the facial bones. The advantages of the finely controlled adjustments of this instrument are such that a description of it seems advisable.



An instrument for anchoring fractures of bones of the head.

By reference to the accompanying figure, one sees that the instrument is comprised of a metal band curved to conform to the head and slotted to take the U clamp pivot bolt. Attached to each U clamp by adjustable blocks is a resilient wire, bent and protected by rubber so that it can be used in this instance as a nasal splint for raising the bridge of the nose. Thumb nuts when tightened lock the U clamp to the metal band and the wire splint to the U bolt. The metal band is held firmly in position on the head by a properly fitting plaster cast or an adjustable strap. Other splints may be used with the instrument, for it lends itself readily to a large variety of traction and pressure rigs. Carefully controlled adjustment is had, enabling force to be exerted in any direction, not only at the time the apparatus is applied but during the course of healing. The care and the delicacy with which the degree and the direction of force can be controlled lend greatly to the comfort of the patient. Aggravation of the injury through excessive motion of the parts at the time of adjustment is lessened. The results obtained in fractures of the facial bones are therefore improved, there is greater comfort and the healing period is shortened.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

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PASADENA, CALIF.

The majority of articles relating to otitis media and complications appearing in 1943 were concerned with the use of chemotherapy. Several articles from men in the military services appeared. Editorial comments appear in brackets.

DIAGNOSIS

Nelson [1] emphasizes the importance of an examination of the hearing before diagnosing acute otitis media. Until serum or pus forms, the hearing remains normal, and air conduction remains better than bone conduction. For ten years he has made it a rule never to incise a drum membrane as long as air conduction is better than bone conduction, regardless of redness, pain, fever or apparent bulging. "This rule has not failed me in the past eight years and in over 100 cases has enabled me confidently to avoid surgical intervention when I should otherwise have feared to delay it." [For several years I have carried a small tuning fork in my bag for this test. It is reliable. With patients too young to cooperate with the fork tests, a hearing test by whisper and voice is of value. A patient with normal hearing does not need myringotomy. This is a valuable test in differentiating between true otitis media and myringitis.]

In an attempt to evaluate tenderness over the mastoid process Rosenwasser [2] warns against being misled by hypersensitiveness of the patient, presence of an inflamed lymph gland over the mastoid process or furunculosis occurring during otitis media. [During internship the reviewer and an attending surgeon were deceived by the swelling back of the ear caused by a beginning furuncle in a child who had had a draining ear for three weeks. Operation, done without waiting for a roentgenogram, showed a perfectly normal mastoid process.] Rosenwasser also mentions the masking of tenderness accompanying chemotherapy. He quotes Friesner as stating that roentgen therapy would make tenderness less noticeable for a day or two.

CHEMOTHERAPY

Furstenburg [3] agrees with the majority opinion that the local application of sulfonamide drugs in infected wounds is of value. For five years the wounds of simple and radical mastoidectomy have been treated with a sulfonamide compound. At first sulfathiazole was used, but for this sulfanilamide was substituted because of its greater solubility. He opposes the use of sulfonamide compounds in clean wounds, where subsequent infection is not anticipated, because of its tendency to produce hemorrhage and delay healing. At his clinic the cavities from simple and radical mastoidectomy are packed full of sulfanilamide powder. The cavity from simple mastoidectomy is closed tight except for a small wick drain at the lower extremity of the wound. The cavity from radical mastoidectomy is filled completely with only a pack for the external meatus. Furstenburg urges that dressings be done rarely and that once the wick is removed it be not replaced, because of the proved tendency to contaminate the wound. "Wounds tend to heal more kindly, more rapidly and with less discomfort to the patient when the sulfonamides are used." Confirming Furstenburg's remark on hemorrhage, Galloway [4] says that there is a tendency for sulfathiazole to cause "troublesome hemorrhage when the microcrystals are blown between the flaps after a submucous resection." Walter H. Theobald in the same discussion mentions that in a series of cases of tonsillectomy the tonsillar fossa treated with "sulfonamides" had more bleeding than the control.

1. Nelson, R. F.: Exact Diagnosis of Otitis Media, Arch. Otolaryng. **37**:810 (June) 1943.

2. Rosenwasser, H.: Clinical Evaluation of Tenderness of the Mastoid, Arch. Otolaryng. **38**:447 (Nov.) 1943.

3. Furstenburg, A. C.: Local Sulfonamide Therapy in Otolaryngology, Laryngoscope **53**:93 (Feb.) 1943.

4. Galloway, T., in discussion on Livingston, G. S.: Clinical Experience with Sulfonamide Compounds in Otolaryngologic Practice, Arch. Otolaryng. **37**:603 (April) 1943.

Livingston [5] gives a résumé of his experience with sulfonamide compounds. He reports 13 cases in which the wound from mastoidectomy was treated with sulfathiazole (5 cases) and sulfanilamide (8 cases) and then closed without drainage. In 11 cases the wound healed by first intention and the middle ear was dry in an average of six and four-tenths days. In 2 cases in which the wound burst open there was a great quantity of serous drainage, probably "due to an excessive quantity of drug in the wound." Sulfadiazine was the choice of the sulfonamide compounds to be given by mouth, and the dosage for children was 1 grain (0.06 Gm.) per pound of weight in twenty-four hours. If sepsis or meningitis was present, a twenty-four hour requirement was given orally as a first dose, or was given by vein as a 5 per cent solution of sodium sulfadiazine in distilled water. In order to "sound a note of caution against their promiscuous and needless administration," he cites 10 cases from the literature in which death from damage of the kidneys occurred.

Marcotte [6] reports cases in which sulfanilamide was used in the wound from mastoidectomy. Complete closure was not used. The time of healing was reduced 50 per cent. In every case the wound was dry in ten days.

Fenton [7] prefers sulfathiazole powder for local treatments. Over a period of eighteen months he has used complete closure with no bad results.

Bowers [8] reports that in the past three years he has used chemotherapy in 50 per cent of his 793 cases of acute otitis media. The incidence of mastoidectomy was reduced about 50 per cent.

In local treatment of chronic otitis media, Lawson [9] used a suspension of sulfanilamide (1 Gm. in 30 cc. of distilled water), which the patient instilled in the ear at home. Careful cleansing of the ear must be done from time to time by the otologist. Ten cases are reported in detail in which cure was obtained after chronic drainage lasting for as long as sixty-six years. In several of his cases granulations and polypi were observed to disappear, and in 2 cases an old large perforation was observed to heal with resulting improvement in hearing. He did not obtain as good results with the powder as with the suspension. Sketches of the drum membranes showed the size of the perforations which healed. [This enthusiastic report will require confirmation.]

Cunning,[10] in a résumé giving the technic of employing chemotherapy at the Manhattan Eye, Ear, and Throat Hospital, New York, states that sulfadiazine is the medicament of choice. He advises that a solution of sodium sulfadiazine be injected intravenously at the start in cases of meningitis. The blood level is kept up to 15 to 20 mg. per hundred cubic centimeters, and in 2 cases of Pneumococcus type III meningitis the concentration was kept up to 40 and 45 mg. per hundred cubic centimeters.

The incidence of renal complications, the most common toxic reaction, can be cut down greatly by forcing fluids and alkalizing the urine. If there is no improvement in three days, he advises stopping administration of the drug. A blood count, a determination of the concentration in the blood and urinalysis should be done every forty-eight hours.

The unfavorable report by Lindsay and Judd [11] on the local use of sulfonamide compounds and tyrothricin in sinusitis does not contradict the foregoing opinions. They report favorable results in using sulfathiazole as a dusting powder in sinus cavities after radical operations but not in local treatment of sinuses in which the mucosa is present. Careful, prolonged treatment of sinuses by irrigation with different solutions, the displacement technic and sprays produced no definite improvement attributable to the drugs. This opinion is confirmed by Walsh, Boies and Williams.[12]

Crowe and co-workers [13] summarize results obtained in treating with penicillin and tyrothricin in 118 cases. Titration of the drugs with the organisms was done to decide to which medication the organism was sensitive. "One of the

5. Livingston, G. S.: Clinical Experience with Sulfonamides in Otolaryngology, Ann. Otol., Rhin. & Laryng. **52**:171 (March) 1943.

6. Marcotte, R. H.: Sulfanilamide Implantation in Mastoid Wounds, Ann. Otol., Rhin. & Laryng. **52**:126 (March) 1943.

7. Fenton, R.: Local Use of Sulfathiazole in Otolaryngologic Practice, Arch. Otolaryng. **37**:491 (April) 1943.

8. Bowers, W. C.: Present-Day Treatment of Infections of Middle Ear and Nasal Sinuses, Eye, Ear, Nose & Throat Monthly **22**:293 (Aug.) 1943.

9. Lawson, L. J.: Chronic Suppurative Otitis Media, Arch. Otolaryng. **38**:550 (Dec.) 1943.

10. Cunning, D.: Chemotherapy in Nose and Throat Diseases, Ann. Otol., Rhin. & Laryng. **52**:394 (June) 1943.

11. Lindsay, J. R., and Judd, D. K.: Observations on Local Use of Sulfonamides and Tyrothricin in Upper Respiratory Tract, Tr. Am. Acad. Ophth. (1942) **42**: 431 (July-Aug.) 1943.

12. Walsh, T.; Boies, L., and Williams, H., in discussion on Lindsay and Judd,[11] p. 439.

13. Crowe, S. J.; Fisher, A. M.; Ward, A. T., Jr., and Foley, M. K.: Penicillin and Tyrothricin in Otolaryngology, Ann. Otol., Rhin. & Laryng. **52**:541 (Sept.) 1943.

reasons of failure in treatment of infections is failure of the clinician to make routine aerobic and anaerobic cultures. . . . The sulfonamides given by mouth or used locally are of no value in infections due to the anaerobic streptococcus." They decided that tyrothricin used as drops or in irrigation of chronically infected sinuses or ears is of little value. Penicillin, because of its deeper penetration, is more valuable.

The technic for testing the sensitivity of bacteria to penicillin and tyrothricin is given. This sensitivity varies greatly with the different strains of organisms. [The case reports and tables deserve study.]

MASKING

Rubin, Sanger and McBean [14] report 7 interesting cases observed at Fort Sill, Okla. All showed a "masking" effect of the sulfonamide compound used in medication. Their first patient received 60 grains (3.88 Gm.) of sulfathiazole for three days. As he looked and felt well, medication was stopped. On the tenth day he suddenly presented symptoms of meningitis and died seventeen hours later. The organism was Pneumococcus type I. In their second patient thrombosis of a lateral sinus developed on the sixth day in spite of 44 grains (2.85 Gm.) of sulfathiazole daily. The organism was Pneumococcus type III. Recovery followed removal of the clot without ligation. Their other 5 patients whose cases were reported showed "masking" of symptoms with dosages of 45 grains (2.9 Gm.) daily. [Most authors would consider this dosage inadequate. Masking of symptoms without cure of the disease is more likely to occur with inadequate therapy.]

Coleman [15] reports 10 cases seen at the Manhattan Eye, Ear, and Throat Hospital. All had serious intracranial complications due to inadequate medication with sulfonamide drugs and inadequate observation. He emphasizes that the medication must be kept up after the infection has apparently subsided and quotes Law, who states that the administration of sulfonamide drugs alters the appearance of the roentgenograms to show less pathologic change than is really present.

Parsell [16] reports a case in which there was masking of symptoms and gives an outline of treatment of otitis media in children.

Gilmore [17] states that the picture of "masked" mastoiditis is almost identical with that of infection produced by Pneumococcus type III. He quotes Converse as stating that animals fail to form antibodies protective against streptococcic infections if treated with sulfanilamide. [This is doubtful. Evidence indicates immunity is formed while sulfonamide drugs are being given. But in too many cases they are not given long enough for enough immunity to develop before the drug is withdrawn.]

Theodore Walsh [18] enters a plea against the use of the term "masking."

> The whole picture of otitis media and mastoiditis has changed in these last years. One can no longer rely on the old textbook signs of mastoiditis as they were before the advent of chemotherapy. One must now learn the signs and symptoms of the otitis media–sulfonamide compound syndrome. . . . Consideration of the possibility of signs being masked by sulfonamide compounds might lead to these agents being withheld, which I think would be a great pity.

[This is a good point, but "masking" is a term now generally accepted; it would be hard to find a better one.]

ACUTE OTITIS MEDIA AND MASTOIDITIS

Connor [19] stresses the point that retention of secretion is the central factor in acute otitis media, and the earlier the drainage, the more effective will it be in controlling the course of the suppuration. He thinks that, for the same reason, chemotherapy should also be used early. [Is it not also possible that the control of otitis media with sulfonamide drugs without incision may help to promote adhesive inflammatory changes ending in impaired hearing?]

Woodson [20] states that "with persistent pain following the incision of an ear drum we may be sure of the onset of a severe fulminating infection." He gives several case histories and advises early chemotherapy and mastoidectomy later. [Severe pain and marked deafness after myringotomy are symptoms to be regarded seriously. Patients with these symptoms should have adequate chemotherapy and careful observation.]

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14. Rubin, H. G.; Sanger, W. W., and McBean, J. B.: Otitic Complications and the Sulfonamides, Ohio State M. J. **39**:125 (Feb.) 1943.

15. Coleman, L. L.: Masking Effects of the Sulfonamides, Ann. Otol., Rhin. & Laryng. **52**:694 (Sept.) 1943.

16. Parsell, L. A.: Acute Mastoiditis in Children, Eye, Ear, Nose & Throat Monthly **22**:266 (July) 1943.

17. Gilmore, G. B.: Acute Mastoiditis Masked by Treatment with Sulfonamide Compounds: Clinicopathologic Consideration, Arch. Otolaryng. **37**:785 (June) 1943.

18. Walsh, T. E., in discussion on Bois, L. R.: Chemotherapy, Arch. Otolaryng. **38**:535 (Nov.) 1943.

19. Connor, C. E.: Acute Suppurative Otitis Media, Minnesota Med. **26**:257 (March) 1943.

20. Woodson, B.: An Evaluation of Some Early Otogenic Complications, South. M. J. **36**:486 (July) 1943.

ACUTE CATARRHAL OTITIS MEDIA

Hantman [21] reports 76 cases of otitis media "due to a low grade or hypovirulent infection of the eustachian tube and middle ear." Several beautiful color photographs are reproduced, clearly showing the typical yellowish fluid level. He properly differentiates this condition from aero-otitis media, in which myringotomy should not be done. His routine treatment in the cases in which the otitis did not clear with a few inflations was myringotomy followed by inflation. In 3 cases suppuration developed.

[In the majority of cases secretory otitis media will clear up eventually without incision of the drum membrane. I have followed a number of patients with this type of otitis media through recurring attacks separated by as long as ten year intervals. All except 1 were treated by inflation without incision. No permanent loss of hearing was noted with one possible exception. When early restoration to duty is important, incision followed by inflation is the method of choice.]

Hoople and Blaisdell [22] state that 666 patients of their 804 with acute catarrhal otitis media had infections of nose and throat just before the onset of aural symptoms. Of the 100 patients with no infection, 68 had otitis media due to allergy, 19 to hypothyroidism, 2 to pregnancy, 7 to aero-otitis, 2 to trauma and 2 to hypopituitarism. Incision of the drum membrane and inflation were done in all cases in which fluid was present, about 180 cases. The otitis media which was due to hypothyroidism cleared up rapidly after administration of thyroid extract.

CHRONIC OTITIS MEDIA AND MASTOIDITIS

Towson [23] analyzes the data on 526 patients with mastoiditis, acute and chronic, seen in a period of ten years in one hospital. The mortality was 5.8 per cent. The incidence of intracranial complications was 19 per cent in cases of acute and 20 per cent in cases of chronic mastoiditis. Almost 50 per cent of patients with chronic mastoiditis had cholesteatoma.

There are 27 tables analyzing different elements of the problem. Nearly 50 per cent of the patients with chronic otitis media showed cholesteatoma; 98 per cent of the patients with acute surgical mastoiditis had dry ears, and 90 per cent of those who had undergone radical operations had dry cavities.

Hubert [24] reports a case of chronic otitis media with probable cholesteatoma in which Horner's syndrome was noted. Radical mastoidectomy had been done, but drainage persisted. No other cause could be found for the drooping right eyelid and the constriction of the right pupil. The patient refused reoperation. No similar case was recorded in the American literature, but several were found in the German and the French literature.

Hubert states that at the Manhattan Eye, Ear, and Throat Hospital he frequently sees patients with paralysis of the cervical sympathetic system resulting in Horner's syndrome. One of the causes of the syndrome is a malignant tumor in the nasopharynx. By instillation of a solution of epinephrine hydrochloride into the affected eye one can tell with reasonable certainty whether the lesion is (1) in the superior cervical ganglion and its postganglionic fibers or (2) in the preganglionic fibers and the central nervous system. In the first case the pupil will dilate, the drooping of the eyelid will disappear and the eyeball will protrude slightly. This reaction occurred in the case reported. The test may require an hour or longer before reaction occurs; the darker the pupil, the longer it takes to get the reaction.

Conley [25] presents an analysis of the data on 100 patients admitted to an Army general hospital. Many of them were sent in from station hospitals. Of the 59 patients with chronic otitis media, 20 showed clearing of the involvement on local treatment. Sulfathiazole powder was found unsatisfactory because of caking and hardening. Thirty-three patients required radical operations.

In the discussion of this paper Horace Williams states that he considers the local application of sulfonamide compounds to be the best local treatment for chronic otitis media.

Emerson and Dowdy [26] report treating 30 patients with radium whose chronic otorrhea appeared, on examination, to be due to hypertrophied lymphoid tissue in and about the eustachian orifice. The report comes from the depart-

21. Hantman, I.: Secretory Otitis Media, Arch. Otolaryng. **38**:561 (Dec.) 1943.

22. Hoople, G. D., and Blaisdell, I. H.: The Problem of Acute Catarrhal Otitis Media, Ann. Otol., Rhin. & Laryng. **52**:359 (June) 1943.

23. Towson, C. E.: Acute and Chronic Mastoiditis: Clinical Analysis of 526 Consecutive Operations, Arch. Otolaryng. **38**:32 (July) 1943.

24. Hubert, L.: Horner's Syndrome with Chronic Purulent Otitis Media: Demonstration of a Diagnostic Test, Laryngoscope **53**:47 (Jan.) 1943.

25. Conley, J. J.: Analysis of One Hundred Consecutive Cases of Aural Disease in an Army General Hospital, Arch. Otolaryng. **38**:541 (Dec.) 1943.

26. Emerson, E. B., Jr., and Dowdy, A. H.: Radium Therapy as an Aid in the Control of Chronic Otorrhoea, Am. J. Roentgenol. **49**:234 (Feb.) 1943.

ment of radiology of the University of Rochester. Patients showing polypi of the middle ear, caries of the ossicles or signs of mastoiditis are not included. The patients were examined with the nasopharyngoscope. Preexistent infection in sinuses, tonsils and adenoids must be treated first. The ages of their 30 patients were from 6 to 25 years. The period of drainage had varied from five months to twenty-five years, averaging perhaps ten years. In most of the cases the infection cleared up in a few weeks. The authors do not offer this treatment as a cure for all otitis media but think that in carefully selected cases the method has definite value.

PETROSITIS

Jones [27] reports a case of petrositis and meningitis caused by Pneumococcus type III. Sulfadiazine was forced until there was a blood level of 45 mg. per hundred cubic centimeters. It is interesting to note that the spinal fluid level was 35 mg. at the time that the blood level was 45 mg. The patient recovered.

Tobey, Mysel and Halton [28] report 2 cases of petrositis with recovery.

THROMBOSIS OF THE LATERAL SINUS

Adler and Klepper [29] report a case of thrombosis of the lateral sinus in which the organism was Bacillus proteus. In spite of heavy doses of sulfadiazine, sulfathiazole and sulfapyridine and ligation of the jugular vein, the patient died. Autopsy showed pulmonary abscesses and empyema.

Proctor, Lindsay and Gonne [30] report 4 cases of sequestration of the osseous labyrinth. Two of the patients were children, and the sequestration occurred in them following scarlet fever and petrositis. The other 2 were adults, 1 with chronic perilabyrinthine petrositis and 1 with diabetes. Pain and vertigo were prominent symptoms. In 3 of the 4 patients complete facial paralysis developed and the fourth had paralysis temporarily.

In 1 patient meningitis developed. This patient recovered after operation and chemotherapy. One patient died of a cerebellar abscess. In 1 instance roentgen examination showed the sequestrum before operation. In all 4 cases the hearing was completely destroyed. Petrosal suppuration is stressed as a predisposing factor. [Necrosis of the petrous bone is to be expected frequently in patients with diabetes, probably as a result of the presence of infected thrombi in the arteriosclerotic vessels found universally in patients with diabetes.]

MENINGITIS AND ABSCESS OF THE BRAIN

Frank [31] reports a case in which symptoms of meningitis began two days after incision of the drum membrane. There was facial paralysis. Sulfapyridine was given in large doses at the time, with apparent recovery. Culture of the turbid spinal fluid showed Pneumococcus (not typed).

The ear still discharged, and six weeks later there was a recurrence of the meningitis. Radical mastoidectomy and drainage of the labyrinth by Neumann's method were done. A fistula was observed in the lateral semicircular canal. Recovery followed prolonged sulfanilamide therapy. The facial paralysis was clearing six months later.

Ballenger, Schall and Smith [32] observed 5 patients with Bacteroides meningitis in five years at the Massachusetts Eye and Ear Infirmary, in Boston; 4 recovered. Only 11 cases of meningitis due to this germ have been reported in the past forty years. The rarity of the reported cases is due to the failure to make routine anaerobic cultures. Sulfadiazine probably was of value in the treatment of the patients. Injections of penicillin and transfusions of immune blood were also used.

TECHNIC

Lathrop [33] reports 15 cases in which the endaural approach was used to perform the modified radical mastoidectomy. In 6 cases the hearing was improved; in 7, unchanged, and in 2, reduced. The observations in the 15 cases are summarized, and the technic is outlined. Better exposure is obtained by the endaural than by the old postauricular route, and results are definitely better.

27. Jones, M. F.: Petrositis and Meningitis with Recovery, Ann. Otol., Rhin. & Laryng. **52**:181 (March) 1943.

28. Tobey, H. G.; Mysel, P., and Halton, E. J.: Mucocele of Frontal Sinus: Report of Three Cases, Arch. Otolaryng. **38**:89 (July) 1943.

29. Adler, H. M., and Klepper, C.: Bacillus Proteus Septicemia Accompanying Acute Mastoiditis with Thrombosis of the Lateral Sinus, Arch. Otolaryng. **37**: 74 (Jan.) 1943.

30. Proctor, B.; Lindsay, J. R., and Gonne, W. S.: Sequestration of the Osseus Labyrinth, Arch. Otolaryng. **37**:819 (June) 1943.

31. Frank, T. J. F.: Pneumococcal Meningitis of Otitic Origin: Recovery Following Chemotherapy and Operation, M. J. Australia **1**:571 (June 6) 1943.

32. Ballenger, J. J.; Schall, L. A., and Smith, W. E.: Bacteroides Meningitis: Report of Case with Recovery, Ann. Otol., Rhin. & Laryng. **52**:895 (Dec.) 1943.

33. Lathrop, F. D.: Endaural Atticomastoidectomy: An Evaluation, Tr. Am. Laryng., Rhin. & Otol. Soc. **49**:236, 1943.

Conley,[25] already quoted under "Chronic Otitis Media and Mastoiditis," reports that mastoidectomy in the majority of his cases in which the operation was necessary was done by the endaural route.

MISCELLANEOUS

Berger [34] reports the case of a woman of 26 who had had drainage from one ear since childhood. Cholesteatoma had destroyed the posterior wall of the external canal and the lateral sinus plate, allowing the lateral sinus to protrude into the canal. The patient was treated by cleansing the canal and insufflating sulfanilamide powder. No other case was found recorded in the literature.

Altmann and Waltner [35] report the case of a woman of 35 who had had a chronic discharge of the left ear for twenty-seven years. The onset followed mumps. She died of an abscess of the temporal lobe, and careful postmortem examination indicated that the cholesteatoma started in the canal rather than in the middle ear. The authors assume that skin had grown into a cavity formed by sequestration of a superficial area of the bony wall of the canal.

34. Berger, I.: Cholesteatomatous Exposure of the Sigmoid Sinus Through the Posterior Osseous Canal Wall, Arch. Otolaryng. **37**:565 (April) 1943.

35. Altmann, F., and Waltner, J. G.: Cholesteatoma of the External Auditory Meatus, Arch. Otolaryng. **38**:236 (Aug.) 1943.

Abstracts from Current Literature

Ear

THE EFFECT OF HEAD INJURY ON HEARING. W. E. GROVE, J. Speech Disorders **8**:363 (Dec.) 1943.

Lesions resulting in defective hearing are restricted to the eighth nerve or to the cochlear contents. The results of an examination for hearing in the early days after injury are not reliable, owing to the disturbed condition of the sensorium. Repeated examinations are necessary in each case for medical or legal purposes, for the detection of malingering. A history of the case should be elicited. Two hundred and eleven cases of proved fracture of the skull were studied, and 49 instances of fracture involving the base of the skull. There was perceptive deafness in 45 cases. In cases of unilateral longitudinal fracture the function of hearing is more damaged as age increases. The perceptive type of deafness is most common. The ear on the side of the fracture sometimes shows a greater loss of hearing than its fellow, but in a large number of instances the loss is the same in both ears. In 65 per cent of the cases of bilateral longitudinal fracture there was loss in perceptive hearing. Labyrinthine fracture produced total deafness on the affected side in every instance and some loss on the opposite in most instances. One person had complete loss of hearing as a result of labyrinthine fracture. Patients may regain a great deal of hearing even a month after an accident. If deterioration of hearing does not occur in the first six months, the condition will probably remain stationary.

The author discusses the methods recommended by the American Medical Association for evaluating loss of hearing. These methods probably will not be useful in evaluating the amount of loss suffered by men in the armed forces because of the method in which hearing is tested at the time of induction.

PALMER, Wichita, Kan. [AM. J. DIS. CHILD.]

RADIATION OF THE EAR. H. B. PERLMAN, Laryngoscope **54**:255 (June) 1944.

Perlman observed the results of irradiation of (1) normal ears, (2) ears that are pathologic as a result of carcinoma of the nasopharynx obstructing the eustachian tube, and (3) ears with a clean perforation of the cochlea (in order to determine the effect on bone repair).

Patients were exposed to 15 roentgens (r) per minute and 300 r per day. Their total dose of radiation was from 3,300 to 5,900 r. No detrimental effect was noticed in appearance or function. In rabbits there also was no detrimental effect on cochlear function (measured by the acoustic tensor tympani reflex) nor could any be detected by microscopic sections of the labyrinths.

There was no detrimental effect of similar irradiation of ears affected by carcinoma of the nasopharynx. On the other hand, in these ears the hearing, the appearance of the drum membrane and (when made) the tubal function all returned to normal.

The effect of 1,500 to 5,000 r of radiation was studied on the rate of healing of a clean perforation made surgically in the cochlea in rabbits. The radiation delayed the healing but did not arrest it. No other effect was noted.

HITSCHLER, Philadelphia.

Larynx

HEMOPHILUS INFLUENZAE TYPE B LARYNGITIS WITH BACTEREMIA. P. G. DUBOIS and C. ANDERSON ALDRICH, J. Pediat. **23**:184 (Aug.) 1943.

Four cases of laryngitis and bacteremia caused by Haemophilus influenzae type B are reported. The clinical picture first described by Sinclair is emphasized because with it in mind the diagnosis may usually be made on inspection. The condition is characterized by (*a*) acute onset, (*b*) extreme prostration or "shock" out of proportion to the local symptoms, (*c*) severe laryngitis with extreme edema and hyperemia of both the false and true cords, (*d*) fever and (*e*) definite neutrophilic leukocytosis. The diagnosis is made accurately when H. influenzae is demonstrated in cultures of blood and of material from the larynx. Vigorous treatment with sulfonamide drugs after prompt tracheotomy was followed by early recovery in 3 of the 4 patients. The fourth patient recovered after a stormy, complicated course. Tracheotomy is advised because most of the patients are exhausted to the point of collapse, and the surgical procedure seems to decrease the severity of the disease and to shorten its course.

SONTAG, Yellow Springs, Ohio.
[AM. J. DIS. CHILD.]

THE ESOPHAGOPHARYNX, A SPECIAL AREA OF THE HUMAN PHARYNX. THOMAS HORACE EVANS, Laryngoscope **54**:148 (March) 1944.

Evans points out that there is another area of the pharynx: the esophagopharynx. It is an area already recognized in comparative literature. The esophagopharynx is the distal end of the pharynx, at the upper end of the esophagus. Its activity is "chiefly to support the upper ostium of the esophagus against which the bolus is forced and to close down in a sphincteric manner on the bolus as it is received within the tube of the esophagus." The muscles constituting this area and their innervation are described by the author.

HITSCHLER, Philadelphia.

Nose

SULFADIAZINE IN THE TREATMENT OF TWO HUNDRED CASES OF ACUTE CATARRHAL NASOPHARYNGITIS. F. B. FAUST and J. M. SIMMONS, J. A. M. A. **125**: 552 (June 24) 1944.

"Nasopharyngitis, acute, catarrhal," is the diagnosis of the common cold as officially given by the Medical Corps of the Army of the United States. The authors studied and analyzed a series of 200 cases of acute catarrhal nasopharyngitis, utilizing local and general measures, and isolating the infected persons in order to lessen the incidence of morbidity.

They summarize their ideas and observations as follows: 1. In an Army post prophylaxis against nasopharyngitis can be accomplished by isolation of patients who actually have the disease. 2. In 113 of 200 consecutive cases studied sulfadiazine therapy similar to that used in pneumonia was employed, and 87 cases

were used as controls; the average number of hospital days for both series was exactly the same—5.6 days. 3. A study of the morbidity shows that sulfadiazine will shorten the hospital stay of the more seriously ill patient. 4. Sulfadiazine-treated patients whose throats revealed presence of cocci or diplococci when examined by making bacteriologic smears recovered more rapidly than those showing mixed infections or staphylococci. 5. There was no pronounced difference between the racial groups. 6. Patients in whom complications developed recovered more rapidly with the use of sulfadiazine. 7. Only 1 case of untoward reaction was encountered in this study.

The authors conclude: 1. Sulfadiazine judiciously used in the treatment of nasopharyngitis is an aid in reducing the number of hospital days for patients with greater morbidity. 2. Sulfadiazine is of definite value in the treatment and the prevention of complications of nasopharyngitis.

GORDON, Philadelphia.

MUCOSAL CYSTS OF THE MAXILLARY SINUS. GERHARD D. STRAUS, Laryngoscope **54**:267 (June) 1944.

Cysts of the maxillary antrum often produce headache. This headache may be of a vague nature and its cause easily overlooked.

Cysts of the maxillary antrum may be of dental origin (dentigerous and radicular cysts) or of mucosal origin (secretory and nonsecretory cysts).

The characteristics and the diagnosis of the cysts are described. It is noted that, although the yellow fluid obtained in the nonsecretory mucosal cyst is sterile, this cyst may act as a focus of infection.

The cysts cause pain, most commonly frontal headache, but often atypical in nature. There is rarely a nasal discharge unless sinusitis is present. Rhinoscopic examination usually does not reveal the condition. Puncture of the antrum may disclose the characteristic fluid. Roentgenograms are of great value in diagnosis. Sometimes roentgenography must be preceded by instillation of iodized poppyseed oil.

Treatment may be simple puncture of the antrum, the creation of an antral window or the Caldwell-Luc operation. The latter is the most satisfactory.

HITSCHLER, Philadelphia.

Miscellaneous

ORAL MONILIASIS IN NEWBORN INFANTS. NINA A. ANDERSON, DOROTHY N. SAGE and E. H. SPAULDING, Am. J. Dis. Child. **67**:450 (June) 1944.

Working in the department of pediatrics of the Temple University Medical School, Philadelphia, the authors say that in nurseries for newborn infants oral moniliasis is exceeded only by impetigo in number of cases. The vagina of the mother is a source, but the condition may spread through a ward, probably via "contacts." Oral thrush developed wherever Monilia (or Candida) albicans was found in the mouths of infants. The fungus persisted long after clinical symptoms had vanished, and therein lies the "carrier" problem! During pregnancy it is important to find the organism and to sterilize the vagina so far as possible. It has been suggested that a newborn infant be kept away from its mother "during the immediate postpartum period." This sounds difficult. Why not cleanse the infant's mouth before and after nursing as well as the mother's nipples? There is no suggestion in the article as to the agent best fitted to destroy the fungus. This is the important desideratum from the point of view of the obstetrician and the pediatrician. Much emphasis is placed on methods of taking cultures from infants' mouths, and seven tables are set down to show the incidence of fungi in the mother's vagina and in the child's mouth. The relationship is self evident. It would seem that the fungus is persistent. One is left in the dark as to the best means of destroying it in either locus.

VOORHEES, New York.

RELATIONSHIP OF POLIOMYELITIS AND TONSILLECTOMY. R. E. HOWARD, Ann. Otol., Rhinol. & Laryng. **53**:15 (March) 1944.

Howard reviews the reports of the relation between poliomyelitis and tonsillectomy. The relationship was first suggested as early as 1910. The organism as a filtrable virus which may be present on any surface of the gastrointestinal tract from the lips to the lower part of the bowel of the person with the disease, the convalescent patient or the carrier. It is widely distributed among normal people but manifests itself only in susceptible ones. The infection may be transferred by hands, handkerchiefs, towels and other recently handled articles of so-called carriers or of persons having the disease. It may also be spread by coughing, sneezing, loud talking or laughing. The recovery of the virus from sewage and flies in infected areas makes one believe that there are indirect methods of spread. The consensus at present is that the virus enters through the mucosa of the alimentary tract.

In his conclusions the author makes the following statements: The possibility of poliomyelitis following removal of tonsils and adenoids during July, August, September and October in nonepidemic years is minimal, but when it does occur it is serious and often of the bulbar type. The ratio is 1 in 2,000 operations. The possibility of the disease following the operation in the other eight months is nil except in Texas and California, where the season for poliomyelitis lasts from June to November and occasionally December. Preventive medicine as practiced now by the boards of health throughout the nation shows that during the months and years when there are epidemics of poliomyelitis, operations on the nose and the throat should be avoided whenever possible.

M. V. MILLER, Philadelphia.

THE ARMY REHABILITATION PROGRAM FOR THE BLIND AND THE DEAFENED. CHARLES C. HILLMAN, J. A. M. A. **125**:321 (June 3) 1944.

Hillman discusses in moderate detail the reports on persons blinded and deafened in World War I and compares those reports with the present ones from various hospitals on similarly afflicted casualties of World War II. He analyzes the rehabilitative measure to be undertaken.

He concludes his otologic report as follows: 1. Deafness in World War II is increased over that in World War I on the basis of figures now available. 2. The Army rehabilitation program for those who have been deafened includes (*a*) treating all deafened persons in three special hospital centers, (*b*) supplying properly fitted hearing aids to all who will benefit by their use and (*c*) offering instruction in residual hearing, lip reading and proper control of speech. 3. Hard of hearing servicemen are salvageable for many types of specialized duty.

GORDON, Philadelphia.

EFFECTS OF CERTAIN DRUGS UPON THE CILIARY ACTIVITY OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY TRACT. D. M. LIERLE and LORANCE B. EVERS, Laryngoscope **54**:176 (April) 1944.

The authors have shown the effects of various widely used drugs and of some of the newer ones on the ciliary action of extirpated nasal mucous membrane. They noted whether the ciliary action was slowed or stopped and the length of time required. They also noted whether the cilia recovered after the tissue had been washed in Locke's solution or in isotonic solution of sodium chloride.

Fresh ephedrine sulfate caused less change in the ciliary action than that made up with chlorobutanol. Many of the solutions of sulfonamide compounds had deleterious effects, while the powders were harmless. Two new solutions (tuamine [2-aminoheptane sulfate] and fouramine [2-amino-4-methylhexane sulfate]) were comparable to ephedrine sulfate when used in proper strengths.

HITSCHLER, Philadelphia.

INHALATION THERAPY USING MINERAL WATERS AND MEDICATED OILS. WALTER S. MCCLELLAN and MARGARET ROGERS, New York State J. Med. **44**:1214 (June 1) 1944.

McClellan is director of a state institution for hydrotherapy. He studied the effects of inhalation of the various waters with which the region abounds on 738 patients. The conditions, variously named, were all generalized infections of the entire respiratory tract; most of them were acute; some were chronic, such as "allergic rhinitis and hay fever." The patients inhaled finely nebulized alkaline carbonated mineral waters and medicated oils. Marked relief was seen in 38 patients, or 5.2 per cent of the total number; moderate relief in 468, or 63.4 per cent; temporary relief in 46, or 6.4 per cent, and no change in 185, or 25 per cent. Relief depended largely on the number of treatments. Patients with acute disease required from four to six treatments; those with chronic conditions, from twelve to fifteen treatments. The author states that there was no discomfort from any of the treatments. He also stresses the safety of the method, since only 3 patients showed any untoward reactions. Of these "one may have had a sensitivity to chlorenan (1 per cent chlorobutanol, 1 per cent menthol and 2.5 per cent camphor in a special solution), one developed an acute asthmatic paroxysm, and one noted a general reaction to epinephrine." He concludes that "inhalations have a definite place in the general 'cure' regimen of a spa." Seven references are listed from the literature. The case histories cited in support of this treatment might have been better evidence if a more thorough "work-up" of the clinical and laboratory agenda had been supplied. In this sense the "'study" lacks scientific value.

VOORHEES, New York.

News and Comment

THE SCIENTIFIC EXHIBIT, AMERICAN MEDICAL ASSOCIATION, PHILADELPHIA SESSION

Application blanks for space in the Scientific Exhibit at the Philadelphia Session of the American Medical Association, June 18-22, 1945, are now available. Applications close on Feb. 12, 1945. The representative to the Scientific Exhibit from the Section on Dermatology and Syphilology is Dr. Hamilton Montgomery, 102 Second Avenue, Southwest, Rochester, Minn.

Requests for application blanks may be addressed to him or to the Director, the Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago 10.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

At the meeting of the Eastern Pennsylvania Association of Eye, Ear, Nose and Throat Physicians in Wilkes-Barre, Pa., May 10, 1944, it was decided unanimously to change the name of the organization to the Pennsylvania Academy of Ophthalmology and Otolaryngology.

The newly elected officers of the organization will be found in the Directory.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY, AND THE PHILADELPHIA LARYNGOLOGICAL SOCIETY

GABRIEL TUCKER, M.D., *Chairman*

LOUIS J. BURNS, M.D., *President*

WILLIAM HEWSON, M.D., *Clerk*

THOMAS F. FURLONG JR., M.D., *Secretary*

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Regular Meeting, March 15, 1944

Vertical Nystagmus Produced by Peripheral Labyrinthine Lesions. Presented by DR. ERNEST A. SPIEGEL (by invitation).

This article was published in full in the ARCHIVES OF OTOLARYNGOLOGY (**40**:160-163 [Sept.] 1944).

DISCUSSION

DR. BENJAMIN H. SHUSTER: In order to maintain continuity of thought in my discussion, it is well for me to summarize the points in Dr. Spiegel's talk. The whole story revolves on the idea that vertical nystagmus observed spontaneously in a patient is thought to be due to an intracranial lesion and that one hardly ever thinks of such nystagmus as being due to a peripheral lesion. Dr. Spiegel brings out by experimental means that such nystagmus can be produced by labyrinthine changes. He tries to prove this by puncturing the round windows of a cat. Vertical nystagmus of a type which changes with the position of the cat's head then developed. If the utricle is impaired, the activity of the vertical canals is thereby increased. In other words, lesions in the two labyrinths under certain circumstances will produce vertical nystagmus.

He also injects into the middle ears a solution of cocaine and on permitting it to act by diffusion he observes certain reactions take place (although more slowly than after puncturing) in which there is vertical nystagmus, and this time the nystagmus is downward instead of upward.

Dr. Spiegel concludes from these experiments that a bilateral peripheral lesion can produce vertical nystagmus.

It is not often that one sees vertical nystagmus due to peripheral lesions. Dr. MacKenzie reported a case in which a focal infection produced vertical nystagmus, which disappeared following extraction of a tooth. I have seen 1 or 2 cases of vertical nystagmus which I thought was of ocular origin.

When Dr. Spiegel considers this subject, he most often discusses it from the experimental angle. I usually think of it in a clinical sense. Clinically I know of no lesion that will destroy bilaterally the round windows and produce the reactions described by Dr. Spiegel.

When a lesion does occur, it is usually a slow process, sometimes taking years to develop, and during this time of pathologic reaction there also takes place a tissue reaction to combat the infection. Erosion, therefore, takes place slowly, and one most often observes the lesions in the horizontal canals. The vertical canals are so deeply placed that the disease hardly gets to them before it is treated clinically.

Experimentally one can produce any type of nystagmus in human subjects at will by rotation. There is nothing new in producing experimental vertical nystagmus. To explain the reactions described by Dr. Spiegel is difficult. One cannot be certain of what takes place in the labyrinth after trauma is inflicted. While this experimental work may be definitely of clinical value in cases in which bilateral lesions occur simultaneously, as a rule, when one sees spontaneous vertical nystagmus, in 99 cases of 100 it is more likely to be caused by an intracranial lesion.

DR. SETH A. BRUMM: It was indeed a privilege to listen to Dr. Spiegel's interesting talk on spontaneous vertical nystagmus due to disease of the end organs. Up to the present it has been believed that spontaneous vertical nystagmus could not occur unless it was due to an intracranial lesion, particularly one in the region of the forward part of the posterior longitudinal tract or, more concisely, one in the region of the deep oculomotor nuclei. Nevertheless, it only proves that medicine continues to be an art and not a science; that is the reason that there is romance in the practice of medicine. In other words, the ports are always open to further research.

For me to discuss this paper "is like bringing coals to Newcastle." Many years ago Dr. Isaac Jones and I did some of the first research relative to the functioning of the labyrinth, and during our work we were able to postulate definite reactions and records which proved to be of practical clinical value.

All are conversant with the fact that nystagmus can be produced in almost any ocular axis simply by placing the head in various planes—for instance, horizontal, vertical, rotary, oblique—and even perverted or so-called compound nystagmus. But the only possible way to produce vertical nystagmus is to place the head in that plane which will stimulate only the anterior vertical canal of one side and the posterior sagittal canal of the opposite side. This physioanatomic certainty makes it rather difficult for me to appreciate Dr. Spiegel's work as having any clinical value. It is therefore difficult for me to visualize lesions that would select only the anterior and posterior canals. Sometimes syphilis will manifest a peculiar selectivity, but even in syphilis I cannot appreciate such refinement that it would be so isolated and circumscript in its destruction. As to Dr. Spiegel's method of producing vertical nystagmus, it was necessary to puncture both vestibules and then to inject the narcotic drug. This again is rather hard to appreciate, as I cannot imagine the anesthetic attacking only the vertical canals. May I say that the only vertical nystagmus which Dr. Jones and I observed when doing our research was observed in those cases in which involvement of the posterior longitudinal bundle was shown? Therefore, I feel that spontaneous vertical nystagmus is still due to an intracranial lesion, and that Dr. Spiegel must present more definite facts to prove his postulations. I hope Dr. Spiegel will continue his excellent efforts, as in science one's most pleasurable rewards are granted when one can disprove old and accepted theories.

DR. ERNEST A. SPIEGEL: I am glad to have this opportunity to discuss the relationship of experimental research to clinical medicine. To deny the significance of animal experiments for the understanding of clinical problems is equivalent to depriving modern medicine of one of its foundations. Bárány's classic work in devising clinical tests for the study of the labyrinth in patients would have been impossible without the physiologic studies of Breuer, Goltz and Ewald, chiefly on pigeons. Many of his clinical rules are simply the application of these experiments on pigeons to the human labyrinth, and most of the recent progress in neuro-otology is owed to the experiments which a pharmacologist, Magnus, and a clinical otologist, de Kleyn, jointly performed on cats. The fact that stimulation of certain vertical canals produces vertical nystagmus is, of course, common knowledge. However, it proves or disproves nothing relating to the present discussion. A superficial study of recent clinical literature already reveals that clinical conditions comparable to my experiments occur. Tamari and also Sommer reported cases of bilateral fistula of the labyrinth in which bilateral compression or aspiration of the air in the external meatus produces vertical nystagmus. Bilateral calorization, introduced by Ruttin, is today a widely used clinical procedure. It has been pointed out by M. H. Fischer that this type of calorization elicits vertical nystagmus when the head is in certain positions, an observation which cannot be explained by isolated stimulation of single semicircular canals but which is easily understandable in the light of my analysis of the macula–semicircular canal relationship. Finally the observation that incomplete paralysis of the two labyrinths may induce vertical nystagmus, as shown in the experiments with cocaine, can never be explained by isolated stimulation of certain semicircular canals. The clinical analogue to this experimental observation seems to be represented by certain cases of toxic bilateral labyrinthitis or labyrinthopathy or bilateral neuritis of the eighth nerve. Dr. Mackenzie's case of vertical nystagmus may belong to this group. Here seems to be a field where cooperation in the laboratory and the clinic may yield further findings.

Cure of Staphylococcic Meningitis of Otitic Origin with Penicillin. Presented by MAJOR WENDELL D. MCCOLLUM, Medical Corps, Army of the United States (by invitation).

Private J. U., a 36 year old white soldier, was admitted to a station hospital Jan. 22, 1944 because of headache, fever and general malaise of twenty-four hours' duration. He had been in excellent health all his life with the exception of the usual childhood diseases and one attack of earache at the age of 5 years, with resultant discharge from the right ear of a few days' duration. Five days prior to admission the patient had an earache on the right side, soon after which he noted a slight discharge. He paid little attention to this and kept on with his duties. The discharge stopped within twenty-four hours, and there were no further symptoms until twenty-four hours before his admission to the hospital.

Physical examination showed a well developed, well nourished man, who appeared acutely ill. The heart, the lungs and the abdomen were normal. The right tympanic membrane showed obliteration of all landmarks. The dried residue of a serosanguineous discharge could be seen in the canal. The vessels of the pharynx were moderately injected. There was slight rigidity of the neck. The blood pressure was 110 systolic and 76 diastolic. The temperature on admission was 102.6 F., the pulse rate 96 and the respiratory rate 24.

A blood count showed red cells 4,780,000; white cells 17,350; hemoglobin 85 per cent. The differential count showed neutrophils 85 per cent, lymphocytes 10 per cent and monocytes 5 per cent.

The urine contained no sugar and no albumin.

The morning of January 23, or approximately twelve hours after his admission, the nurse noted that the patient complained of increasingly severe headache, nausea and stiffness of the neck. Later he vomited. Whether this vomiting was of the projectile type, I do not know. A spinal tap and a blood culture were ordered. A consultation with the Ear, Nose and Throat Service was requested. The patient was seen by Major M. B. Stewart, chief of that service, and his report of his examination follows:

"Right Ear: There is a small perforation of the lower middle part of Shrapnell's membrane. In the canal there remains the dried residue of a serosanguineous discharge. The drum is thickened with no bulging, the landmarks are obliterated, and the color is dark and lusterless. There was no pain in the ear or in the area of the mastoid process, and no tenderness in this area. Impression: Otitis media, suppurative, acute, and meningitis."

A smear of material from the ear showed gram-positive cocci.

The spinal fluid grossly was cloudy. A manometer reading was not obtained, and I am unable to give the pressure. The rate of flow did not indicate any marked increase although, admittedly, this observation may be misleading. In view of the physical findings and the cloudy spinal fluid, the patient was given sulfadiazine without waiting for the laboratory report. He was given 6 Gm. immediately and 2 Gm. thereafter at four hour intervals.

In the early afternoon he appeared more drowsy, but when aroused he was rational. There was profuse diaphoresis.

Myringotomy was performed with resultant serosanguineous discharge. A careful examination of a smear and a culture of the discharge were ordered. The smear revealed gram-positive cocci, later identified as Staphylococcus aureus of a nonhemolytic type.

The report on the spinal fluid was as follows: The smear revealed gram-positive cocci (later identified as Staph. aureus). The cell count showed 5,600 white cells per cubic millimeter, 66 per cent of which were polymorphonuclears and 34 per cent lymphocytes. Globulin was markedly increased. The total protein amounted to 67 mg. per hundred cubic centimeters. The gold curve was 0000000000.

Thirty hours after his admission his temperature had risen to 104 F., the pulse rate to 110 and the respiratory rate to 28. Brudzinski's and Kernig's signs were present, and knee jerks were absent.

In view of the fact that the infective agent was a gram-positive organism, thought to be Staph. aureus, it was realized that sulfadiazine would be inadequate. Penicillin was indicated and its use strongly desired. The evening of January 23 (twenty-eight hours after his admission) treatment with penicillin was initiated. The patient received 10,000 units intramuscularly and 10,000 units intravenously every two hours for a total of 100,000 units during the night.

On January 24 the use of sulfadiazine was discontinued. A spinal tap was done. Cloudy spinal fluid was obtained under a pressure of 250 mm. (water

manometer). Compression of the jugular vein showed an increase to over 400 mm. The temperature was 102.8 F.; the pulse rate, 100; the respiratory rate, 30. The temperature began to rise until it reached a maximum of 105 F. at 1430. It was evident that the penicillin would have to be increased and complemented by intrathecal injections.

The following schedule was initiated: A spinal tap was done daily with removal of 15 cc. or more of spinal fluid. Then 10 cc. of saline solution containing 5,000 units of penicillin was injected into the spinal canal. A continuous intravenous drip of a .5 per cent solution of dextrose in distilled water was arranged, and 1 cc. of penicillin (5,000 units per cubic centimeter) was injected into the tubing of the intravenous drip every half hour. Every three hours 5 cc. of penicillin (25,000 units) was injected intramuscularly. Plasma and blood transfusions were to be given as indicated. Surgical treatment of the ear and mastoid process would be undertaken if indicated.

By the three routes of administration, the patient was receiving a total of 445,000 units of penicillin during each twenty-four hour period.

The temperature began to drop. On January 25 the maximum was 101.5 F.; on January 26, 100.5 F.; on January 28, 99 F. Throughout the remainder of his stay in the hospital the temperature remained normal except for an occasional rise to 99 F.

The daily intrathecal injections were discontinued after the third dose. The intravenous injections were discontinued after four days. The intramuscular injections were discontinued on the tenth day following their initiation. In other words, the patient received a daily total of 5,000 units intrathecally, 240,000 units intravenously and 200,000 units intramuscularly for the three day period during which all three routes of administration were utilized. The total amount of penicillin received amounted to 2,300,000 units.

At the time of his discharge from the hospital, which was four and one-half weeks after his admission, his headaches had disappeared.

The blood cultures were consistently negative throughout the course of the disease. Smears and cultures of the spinal fluid demonstrated the presence of staphylococci in the fluid on January 23 and 24. However, following the first intrathecal administration of 5,000 units of penicillin, on January 24, the spinal fluid obtained the next day showed no organisms either on smears or on culture. It continued to give negative results from then on. The cell count gradually dropped to that of normal spinal fluid within ten days. The eyegrounds were repeatedly examined and found to be normal throughout the course of the disease.

Summary.—This was a case of staphylococcic meningitis secondary to acute suppurative otitis media without any demonstrable involvement of the mastoid process.

The route by which the infection advanced into the subarachnoid space was not determined, but from physical signs and roentgen evidence it was most likely not through the usual area of mastoid cells.

Large doses of penicillin administered simultaneously by intrathecal, intravenous and intramuscular routes brought about a satisfactory and complete recovery.

No beneficial effect was noted from the initial intravenous and intramuscular administration of 100,000 units of penicillin.

Only 15,000 units of penicillin was administered intrathecally, and I do not believe this amount could have been reduced with safety. However, the amounts given intravenously and intramuscularly, totaling 2,285,000, may eventually be proved to have been more than adequate.

No untoward effects were noted from the use of penicillin.

DISCUSSION

DR. ALBERT P. SELTZER: In the Post-Graduate Medical School and Hospital we have a patient who has undergone radical mastoidectomy and exploration for a cerebellar abscess.

Through the aid of the director of public health, Dr. Rufus Reeves, and Dr. Franklin Murphy, we obtained penicillin for her.

It was my impression that the lesion extended in the proximity of the jugular foramen, involving the ninth, tenth and eleventh nerves. The patient had paralysis of the palate with nasal speech, difficulty in swallowing, and hoarseness due to unilateral involvement of the recurrent laryngeal nerve on the side of the lesion.

To date we have given her 700,000 units intramuscularly and intravenously. The Gradenigo syndrome is clearing up but we are keeping a careful watch on her. I hope we shall have the excellent results obtained by Major McCollum.

MAJOR WENDELL D. MCCOLLUM: It is to be regretted that we were not able to get penicillin immediately in the case I discussed. We have penicillin at the hospital but it is only for use in gonorrheal infections that are resistant to sulfonamide drugs. There was, therefore, some delay in administering the penicillin. However, on the second day penicillin was administered, and it did miraculous work.

Fractures of the Malar-Zygomatic Compound and Their Treatment. Presented by MAJOR MARSHALL B. STEWART, Medical Corps, Army of the United States (by invitation).

Fracture of bones of the zygomatic compound is usually accomplished by severe external force. The zygomatic arch is fractured singly more often than the malar bone, 10 to 1. It is therefore understood that the force necessary to fracture the zygoma is much less than that necessary to injure the malar bone. Compound fracture of the zygoma due to any force except that of a weapon is unknown (W. F. Coakley). Fracture of the malar bone may be caused by a direct force such as that encountered in an automobile injury or in a butt of the head. If the maxillary antrum is thin walled, a blow may drive the malar prominence directly into it.

The symptoms and signs of a fracture of the malar-zygomatic compound are: pain; swelling, depression or flatness of the face; trismus if the arch is involved, and limitation of the motion of the jaw because of depression of the zygomatic arch against the coronoid process of the mandible (F. Smith); intranasal hemorrhage on the injured side; diplopia, a most important subjective symptom, caused by displacement of the infraorbital crest; emphysema from fracture of the maxillary antrum and puncture of the antral lining. When the malar fracture extends into the orbit, the depression can be palpated even in the presence of swelling.

There are four sites of fracture, which correspond to the articulations: the frontal process–the superior external orbital margin; the orbital process–the region of the infraorbital foramen; the zygomatic arch; the zygomatic process of the maxilla (F. Smith; Ferris).

Fracture of the zygomatic arch is frequently missed by inexperienced surgeons. Limitation of motion of the

mandible is the most constant symptom of this condition. Displaced fragments can frequently be palpated within and outside the mouth before too much edema is present. Limitation of mandibular motion in the presence of injury should always indicate roentgen examination. The verticosubmental projection will give more diagnostic information in regard to the zygomatic arch than any other.

The method and the time of reduction depend a great deal on the physical condition of the patient and on whether a compound or a simple fracture is present. The reduction of a fracture of the zygomatic arch is a simpler procedure than that of a malar fracture.

The treatment of malar fractures usually demands special planning. Manipulation is more difficult, and fixation and traction are frequently required. The fragments are often impacted, and reduction cannot be delayed as long as with a fracture of the zygomatic arch.

Roberts makes a small incision over the malar prominence and then uses a cork-screw-like instrument for elevation and manipulation.

When the malar bone is detached and movable and the fracture extends into the orbital rim, O'Brien, with the patient under general anesthesia, inserts a blunt instrument intraorally under the malar prominence, and with a gloved thumb inserted in the conjunctival cul-de-sac, rocks the fragment into position.

Fixation of the malar bone can be accomplished in certain cases by direct wiring with tantalum wire. When traction with external fixation is needed, a plaster of paris head cap is applied, a procedure of Ivy and Curtis. A screw eye is inserted through an external stab incision in the malar fragment, which is held in position for about a week.

Repair of associated fractures of the maxilla can be accomplished without a cumbersome plaster head cap, by internal fixation, as described recently by Adams. A small incision is made over the ridge of the frontal bone, lateral to the supraorbital rim, just above the zygomatic-frontal suture line. The periosteum is elevated; a hole is carefully drilled near the rim through the ridge into the orbit (protecting the eye with a retractor). Wire is threaded through the opening and looped around the orbital rim. The two wires are passed through the soft tissues under the temporal surface of the malar bone, the loose bones are elevated into position, and the wires are fastened to the teeth. This holds the maxilla in place and also exerts an outward pull on the malar bone, holding it up in proper position.

The wires can be left in position for several weeks. In order to prevent infection during removal, the original incision of the skin is reopened at the orbital rim, the wire is cut and both wire strands are pulled downward and out through the mouth. This method is revolutionary, quick, simple in application and requires no special apparatus. This new method will supplant the plaster head cap described by Federspiel, in a great many cases.

Complications of a fracture of the malar-zygomatic compound will naturally involve structures such as the maxillary sinus, the nose and the eyeball. Diplopia results from traction on the tendons of the oblique muscles. There may be injury of nerves, especially the infraorbital nerve. The antral wall is frequently crushed, and hematoma in the maxillary sinus is common. Ivy and Curtis do not advise routine drainage of the maxillary antrum for this condition except when suppuration takes place in old fractures with deformity. A cosmetic defect can frequently be corrected by an implant of cartilage or an open type of reduction. Incision across the facial fibers is sometimes necessary, and the patient should be warned in advance of such a complication.

DISCUSSION

CAPT. JOHN J. CONNELLY, Medical Corps, Army of the United States: We suggested to the patient correcting a cosmetic defect with cartilage, and several other refinements which we had planned. He was not impressed, so we did not follow through.

MAJOR MARSHALL B. STEWART: In packing the antrum, iodoform packing was used. It should not be left in more than two days, although it has been left in five days with no discomfort to the patient.

Management of Depressed Nasal Fractures. Presented by DR. HENRY A. MILLER.

"Saddle nose" originally described a characteristic nasal deformity and tertiary syphilis. The term has come into more general use to include any depression in the nasal profile. Such depressions are produced by head on trauma or by loss of septal support following injudicious surgical intervention or by septal infection.

In planning restoration of the depressed nasal contour it is necessary to determine what structures are affected, by careful external and intranasal examination. The depression due to weakening of the septal support by septal infection or by previous surgical treatment produces a sinking-in close to the tip of the nose, while the osseous framework remains normal. Such small defects can be obliterated by the use of a dermal graft or a piece of cartilage inserted under the skin to fill out the defect. Depression of the entire tip with some lateral deviation is due chiefly to a trauma which caused displacement of the septum from its groove between the two nasal spines of the maxillas. It is therefore particularly important that when a child strikes his nose an intranasal examination be carried out. Hematoma of the septum, if present, must be promptly evacuated to prevent infection and destruction of the septum. Displacement of the septal cartilage from its groove, if noted at the time of the accident, can be easily corrected by manual replacement, with the patient under anesthesia. Such a displacement, if unrecognized and allowed to heal, produces progressive depression of the nasal tip, with twist, and prominence of the anterior end of the displaced septum in one of the nostrils. It can be corrected fairly satisfactorily in adult life by a replacement operation such as that described by Metzenbaum or Cohen.

In adults the correction of an old healed traumatic saddle nose will often require first a narrowing of the nose before the correction of the profile is undertaken. This procedure will raise the nasal dorsum to some extent, but it is usually insufficient to correct the defect entirely. The restoration of the profile can be accomplished at the same operation by the use of a cartilage implant, but it is probably safer if done by means of a second operation.

Such cases also require correction of the septum and its attached upper lateral cartilages. It is occasionally feasible to sew the upper lateral cartilages together, back to back, to fill out the depression in this area. In most instances, a cartilage implant is preferable. The septal deformity may require a submucous operation to correct the associated obstruction. When there has been marked displacement of the anterior septum with deviation posteriorly, a Metzenbaum type of operation is necessary to replace the displaced anterior septum and

restore the support of the nasal tip, and a posterior submucous resection is necessary in order to correct the obstruction.

The saddle nose of syphilis is a result of involvement of all the structures of the nose, including the lining. The nose is so bound down that the simple insertion of a piece of cartilage will not suffice. It is necessary to mobilize the entire nose, and the bare areas produced by freeing the nose from its adhesion must be grafted with skin before one undertakes to correct the lost supports and contoural defects. The latter in some cases is managed with cartilage implants and struts, while in others a prosthetic device extending into the floor of the nose or down into the mouth is the only way the nose can be supported in its normal position.

DR. SAMUEL COHEN: I thank Dr. Miller for the privilege of discussing his excellent paper. There are several phases of the subject that I shall stress.

First, in reference to the treatment of new depressed nasal fractures: In general, nasal fractures should be corrected as soon after their occurrence as possible. This dictum is especially true with respect to those of the depressed type. Waiting ten days to two weeks for the swelling to disappear permits excess callus to form and makes the separation, raising and proper alinement of the bony fragments much more difficult and often imperfect. The nasal bones are immediately under the skin and are so conspicuous that any postcorrection is notable and displeasing. Small depressed fractures at the lower ends of the nasal bones are often overlooked. Patients with such fractures give a history of trauma and show slight puffiness over the midnasal dorsum. Superficial examination discloses little. A roentgenogram would demonstrate these small depressed fractures at the lower end of the nasal bones. If these fractures are not treated promptly and correctly, a slight depression develops over the midnasal bridge several months after the occurrence of the trauma. Then it is usually too late to lift the broken ends upward. Therefore, one should make a roentgen study in all cases of nasal trauma.

Dr. Miller spoke of the implantation of cartilage for the correction of saddle nose. The Joseph technic, that, is, implantation through an incision between the upper and lower lateral cartilages and use of the Trelat knife to elevate the adherent skin over the tip, is also a very good method of approach. The nasal vestibule being lined with skin, it can be antisepticized as well as the skin of the columella. A small postoperative vestibular pack used for a day or two prevents the entrance of infection, and an external columellar scar is avoided. However, implantation into the columella cannot be carried out by this approach. To overcome this disadvantage I have used a different intravestibular approach. I presented a short paper on this technic before this society in 1937. The main points of this approach are as follows: An incision is made through the lateral plate of one lower lateral cartilage, parallel to its lower border and extending under the nasal tip. Through this incision a pocket is made upward to the glabella. If a columellar strut is needed, the undermining is continued backward in the columella between the medial plates of both lateral cartilages. One has thus two connecting pockets, one in the columella and one along the nasal dorsum. Both implants can be inserted easily. A small pack is placed in the nasal vestibule, and an external nasal fixation appliance is used.

Retraction of the columella is a frequent and troublesome condition associated with saddle nose. This condition is also frequently seen as a postoperative rhinoplastic complication. I obtained one good result by a novel technic. A small mucosal incision was made under the upper lip at the frenum. With a fine scissors, a pocket was dissected forward in the columella between the two medial plates. A sliver of cartilage was inserted into this pocket. Below this two mattress sutures were placed through the columella. The labial incision was closed by one catgut suture. The mattress sutures were removed in twenty-four hours. The retracted columella is in need of more thorough investigation.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

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Reduced Atmospheric Pressure as a Form of Treatment for Paranasal Sinusitis. Presented by DR. D. B. BUTLER, DR. G. J. GREENWOOD and A. C. IVY, PH.D.

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DISCUSSION

DR. THOMAS GALLOWAY: This study has been made in Dr. Ivy's usual thorough manner. One question of interest is what effect the lowered pressure at higher altitudes has on blood flow and edema in the sinuses. Would it increase congestion by relieving the pressure on the vessels? If so, that is important. If the decreased air pressure is important, there should be other methods of obtaining that effect; if this principle applies, there will undoubtedly be some simpler way. For instance, the patient's head could be placed in a negative pressure chamber. Does displacement drainage by changes of posture account for any of the beneficial effect?

Dr. Ivy uses the term "nontraumatic," apparently in contrast to his idea of office treatment. Yet, suction in the office should not be used to the point of physiologic trauma.

DR. JOHN R. LINDSAY: This has been an interesting presentation. When analyzed, however, the effect of this treatment does not appear to differ from that customarily obtained by other means. Two years ago in the clinic with which I am associated Dr. Judd made a number of observations on the interchange of pressure between the nasal cavity and the maxillary sinus. Normally there is free interchange of pressure at the ostium. In acute and chronic inflammatory conditions there may be a lag in the equalization of pressure, indicating partial blockage at the ostium, which in turn causes retention of secretions in the sinus.

In such cases the use of a vasoconstrictor in the middle meatus opens the ostium, as indicated by a return of free interchange of pressure. The cilia are able to function again, and some mucopus may shortly be expelled into the nose.

It seems that the effect of changes in altitude is similar in that mucopus which may be lodged partly in the ostium is moved into the meatus, thereby removing the impediment to ciliary action.

Both methods probably serve only as aids to ciliary function. The use of alterations in pressure obviates the possible irritating effect of a vasoconstricting agent.

DR. SAMUEL SALINGER: The striking thing about the charts presented by Dr. Ivy is the difference in percentage of improvement when improvement is judged by objective signs instead of subjective symptoms, the percentage with the former being so much less. To my mind this proves what all rhinologists know, namely, that any procedure which assists ventilation of the sinuses is bound to be followed by amelioration of the symptoms. When checked by objective findings, however, one finds that the basic condition responsible for the complaint is still present. It is therefore important in evaluating the report that one know the type of patient receiving the treatment, since it will be shown that certain types respond more readily to simple ventilation than others. It may well be that in Dr. Ivy's group the good results were obtained in only one particular type of sinusitis.

My reaction to the modus operandi of Dr. Ivy's experiment is that the reduced air pressure releases secretion clogging the ostiums of the sinuses, thus permitting air to enter those cavities and restore a normal balance of air pressure, and this in turn facilitates the ciliary activity. I do not believe infected secretion can be sucked into a sinus unless the pressure is great, chiefly because the ciliary wave is constantly in the opposite direction. Such a contention would require more definite experimental proof.

DR. O. E. VAN ALYEA: Dr. Ivy has presented an important series of experiments illustrating problems in physics. To apply it clinically one must go a little deeper. One must attempt to analyze some of the causes of chronic sinusitis. It seems to me that this particular phase of the investigation was not thoroughly carried out. It has been rather well shown that chronic sinusitis does mean poor drainage. I think for proper evaluation it would be necessary to study the ostiums more carefully, as well as the adjacent structures. No mention was made as to the condition of the middle meatus in these cases. What was blocking the ostium—thickened mucosa, polyps, edema of allergy or a deviated septum? I think each case has to be studied thoroughly before one can evaluate the results which Dr. Ivy and his associates obtained. I do not think one can place those cases in a group and establish satisfactory conclusions.

DR. ANDREW C. IVY: I am sorry Dr. Greenwood could not be present. He made the rhinologic examinations and is the one in our group best qualified by experience to answer questions on that subject. As to whether exposure to higher altitude actually caused drainage by suction, it can be said that such suction actually occurred in some but not all patients during exposure. We have some photographs of the maxillary ostiums before and during exposure to high altitude which demonstrate that drainage occurred in some instances. As Dr. Salinger points out, the objective signs of improvement are fewer and less favorable than the subjective ones. However, the disappearance of a long-standing headache without a change in objective findings is significant to us and especially to the patient. We deemed it advisable to ascertain whether clinical subjective improvement could be obtained by the method before expending time and money on the collection of objective data. In time we shall accumulate more objective data. Of course, we doubt whether any amount of therapy will clear organic changes. It is to be recalled that our patients had chronic sinus disorders of long standing. However, as Dr. Salinger points out, the therapy may promote drainage by improving ciliary function. As Dr. Galloway suggests, the improvement may be secondary to hyperemia. In fact, we are finding that when patients are exposed to high altitude, hyperemia, as indicated by inspection and by actual measurement of the resistance to air flow, occurs, regardless of the fact that patients report subjectively that nasal breathing is facilitated, which is probably due to the decreased density of the air.

Regarding the passage of infected material into the eustachian tubes or the sinuses, this might occur if the patient was returned to ground level too suddenly, just as it may occur in an aviator in a power dive or a rapid descent. This might occur if a patient or a subject were placed in a pressure chamber, as was done by Dr. Behnke, and the pressure increased too rapidly.

As suggested, we might obtain better drainage by requesting the patient to place the head in more favorable positions. We have not introduced that factor into the therapeutic procedure. However, gas is in the sinus and in the fluid in the sinus, and its expansion on being exposed to reduced pressure could facilitate drainage during the exposure. However, I suspect that some less obvious factor, such as hyperemia, rather than direct suction drainage operates to produce the improvement observed.

Local Use of Powdered Sulfathiazole in Acute Nasopharyngitis (Common Cold). Presented by DR. HOWARD C. BALLENGER.

The local use of the various sulfonamide compounds in treating acute infections of the upper respiratory tract has been receiving increased attention during the past year, owing largely to the introduction of the soluble sodium sulfathiazole (2.5 per cent) in combination with dl-desoxyephedrine (0.125 per cent). Other combinations and forms of the sulfonamide compounds have been used in the nasal cavities and sinuses, varying from a suspension of the microcrystals (5 per cent) in a 1 per cent aqueous solution of paredrine hydrobromide (parahydroxy-α-methylphenylethylamine) or other solutions or substances, to pure microcrystalline or powdered forms of the drug to be dusted or blown into the nasal cavities or sinuses.

The principal objection to the use of the various solutions is the improbability that they will remain in contact with the nasopharyngeal mucosa long enough to be effective, even though used many times in the course of the day. Another valid objection to the use of solutions of the soluble sodium sulfathiazole is their high alkalinity (p_H approximately 10) compared with the p_H of the nasal mucosa, which is 5.5 to 6.5. However, the p_H of the 2.5 per cent sodium sulfathiazole in the 0.125 per cent vasoconstrictor dl-desoxyephedrine hydrochloride is 8.6 to 9.0, making it still somewhat more alkaline than the nasal mucosa. The powdered or microcrystalline forms of sulfathiazole do not have the objection of excessive alkalinity.

Sulfanilamide and sulfadiazine are much more soluble in the nasal secretions than sulfathiazole, and as a result their time of contact with the nasopharyngeal mucosa would be less than that of sulfathiazole. The sulfonamide compounds, it is generally conceded, have no inhibiting effect on the initiating virus of the common cold or on that of influenza. However, the activated secondary pathogenic bacteria, such as streptococci, pneumococci and staphylococci, which do most of the damage and cause most of the complications, are inhibited in most instances. The sulfonamide compounds are effective, but to a lesser degree, against Haemophilus influenzae and the hemolytic staphylococcus.

During a period of eight months (July 1943 to February 1944, inclusive) approximately 500 attacks of various stages of acute nasopharyngitis were treated in the following manner: An average of about three treatments was given for each attack, although many of the patients had only one treatment. Some patients had two or three attacks during this period.

The nasal cavities were first sprayed with the minimum amount of a 2 per cent aqueous solution of ephedrine hydrochloride to produce sufficient shrinkage of the turbinates and nasal mucosa for a maximum distribution of the powdered sulfathiazole which followed. The powder was blown into the nasal cavities, the nasopharynx and the pharynx until the visible mucosa was covered with a dry chalky white film. During the process of blowing the powder into the nostrils the patient was asked to inhale slowly through the nose. The tip of the powder blower was then turned up and the powder blown behind the palate into the nasopharynx; then, with the tip turned down, a small amount was insufflated into the laryngopharynx. If tonsils were present as much of the sulfathiazole as feasible was blown into the crypts of the tonsils. The patient was asked to refrain from blowing the nose or clearing the throat for as many hours as possible. If the nose was not blown until the next morning, the powder could be demonstrated in the discharge or seen as patches on the nasal mucosa in most instances. Visible evidence of the powder was of much shorter duration in the pharynx than in the nose, irrespective of whether or not the patient refrained from clearing the throat.

The quantity of powder used at each treatment was difficult to estimate, but it was probably much less than 0.3 Gm., as less than 1 pound (453.5 Gm.) of sulfathiazole was used throughout the eight months of treatment. Much of the powder did not adhere to the mucosa but was blown on through or was exhaled.

A presumed sensitivity to sulfathiazole was encountered in 3 patients, as in all 3 a local cutaneous rash developed around the nose or over the face; this disappeared within twenty-four hours. No other discomfort was noted. Any patient with a known or suspected sensitivity to sulfathiazole was not treated with the substance.

An exact evaluation of the clinical results is impossible, but when the therapeutic effects on this series of patients are reviewed and compared with those in an equal number of patients with acute nasopharyngitis treated by other methods, the conclusion is inescapable that the course of the acute infection was shortened and the number of complications was lessened. Further clinical and laboratory investigations are necessary before a satisfactory evaluation of this method of treatment of acute nasopharyngitis can be made.

The objections to the local application of the sulfonamide compounds to the nasopharyngeal mucous membrane would be the same as those to their oral or parenteral use, but possibly less serious. The most important considerations would be the possibility of producing sensitivity, as this has occurred from their use on the skin. However, no sensitization has been noted in this series of cases. If sensitization should occur, even in a very small percentage, the value of the procedure would be limited.

DISCUSSION

DR. J. V. CASSADY: I have used sulfathiazole locally in otitis media and in the antrum after irrigation. I think many patients in whom it has been used promiscuously for common colds have more nasal congestion and more irritation afterward than those who have been using ephedrine or drugs that do not have sulfathiazole in them.

DR. SAMUEL SALINGER: I appreciate and applaud Dr. Ballenger's report because he drew no conclusions. When one considers the various factors that enter into the tabulation of some 500 cases of sore throat and rhinitis, it is extremely difficult to appraise accurately one therapeutic measure without numerous controls, which in this instance were not employed. One must admit that Dr. Ballenger did note that his patients seemed to get well more quickly than had been his experience previously with other measures, and since he failed to see any deleterious effects from the treatment, one must inevitably be impressed.

DR. HOWARD C. BALLENGER: As I said before, the most valid objection to the use of any sulfonamide drug on the nasopharyngeal mucous membrane would be the possibility of sensitization. However, no sensitization has been observed in this group.

Xanthomatosis—Lipoid Granulomatosis of the Temporal Bone. Presented by DR. M. A. GLATT.

A man aged 36 complained of severe left-sided temporal pain, dizziness, deafness and extreme weakness, with no fever, glandular enlargement or discharge from the ear. Roentgen examination showed an area of rarefaction at the temporomandibular joint and the squama, not involving the mastoid cells.

Observation for a period of three months led to the conclusion that the lesion was either a malignant neoplasm or xanthoma. At operation a greenish brown sequestrum was removed which exposed the brain substance. Recovery was uneventful. Histologic studies proved it to be a lipoid granuloma, or xanthoma.

The genesis of this lesion is due either to a disturbance in fat metabolism which produces an infiltration of cholesterol in the reticuloendothelial cells, or to a disturbance of the equilibrium of the fat content within the endothelial cells. The characteristic histologic feature is the presence of "xanthoma or foam cells." This type of granuloma may invade any portion of the skull, including the paranasal sinuses, in solitary or multiple areas. In the temporal bone it may involve the squama, the petrosa and the dura before it progresses to the air cells of the mastoid process.

The symptoms are not typical of ordinary surgical mastoiditis or of malignant growth; nevertheless, clinically the differentiation is not easily made. In the presence of systemic disturbances, known as the Hand-Christian-Schüller syndrome, such as exophthalmos, diabetes insipidus or the finding of other osseous lesions in the skull which show an irregular outline with sharp borders on the roentgen film, the diagnosis of xanthoma should not be difficult. In the presence of a solitary lesion with indefinite borders, diagnosis can be made only on the finding of xanthoma cells in the histologic studies.

It is important to keep in mind the possibility of this lesion in operative cases in which the lesion does not heal well and in which other factors have been ruled out. Surgical procedures should not be undertaken. Roentgen therapy has a favorable influence in producing fibrosis of the xanthomatous lesions.

DISCUSSION

DR. JOHN R. LINDSAY: I treated a patient with a condition similar to that of Dr. Glatt's patient a few years ago. A young man presented himself with a chronic discharge from one ear of several months'

duration, with circumscribed labyrinthitis and an active fistula response. He had in addition a small cyst in the occipital bone on the same side and another in the scapula. Both lungs showed evidence of fibrosis.

A modified radical operation on the mastoid process was followed by complete healing with an intact drum and preservation of function. The labyrinthine fistula healed over. The cysts in the occipital bone and the scapula were also cleaned out. The final diagnosis was xanthomatosis. Until a year or so ago no further difficulties had developed.

DR. THOMAS C. GALLOWAY: I should like to compliment Dr. Glatt on a fine presentation of a difficult subject. I think this disease has been overlooked frequently. My associates and I at County Hospital have seen several cases in recent years, in one of which the involvement was in the frontal bone, involving the sinus, in an infant.

DR. M. A. GLATT: My impression is that in this case the lesion may have started from the dura. There were no other lesions of bone. Xanthoma may involve the bone marrow and produce bone changes, but it is not primarily a disease of the blood-forming organs. Most of the patients do not show any change in the blood picture.

This is a rare disease, but undoubtedly there are cases in which operation has been performed and in which a histologic diagnosis is not made because of spontaneous healing. I feel that histologic study of the removed tissue should be more carefully done. Usually cultures are taken, but tissues are rarely sectioned for study. If this were done, one might find more cases. I should hesitate to apply preoperative aspiration to the skull, where the dura might be involved, as a diagnostic measure. I believe surgical biopsy to be preferable.

Book Reviews

Neurology of the Eye, Ear, Nose and Throat. By E. A. Spiegel, M.D., professor of experimental neurology and head of the department of experimental neurology, Temple University School of Medicine, and I. Sommer, M.D., lecturer in ophthalmology, Long Island College of Medicine, and consultant ophthalmologist and otolaryngologist, Chicago Eye and Ear Hospital. Price $7.50. Pp. 690, with 118 illustrations. New York: Grune & Stratton, Inc., 1944.

This book is an excellent source of information for the advanced student and the practitioner of ophthalmology and otolaryngology. It discusses in an interesting manner all topics in these specialties which in any way are related to the nervous system.

The field it surveys may be discerned from the table of contents. The auditory and vestibular functions of the ear are studied. Ophthalmology in its relations to the central nervous system is thoroughly considered. The neurologic phases of conditions in the nose, the mouth, the pharynx and the larynx are presented, with a discussion of the olfactory tests, the reflexes of pain and other functional disturbances in these regions whose explanation is found in the connection of the terrain with the nervous system.

Finally, a brief discussion of the central nervous system is presented, indicating its relation to the numerous symptoms and manifestations in the eye, the ear, the nose and the throat. While the book was originally conceived as a translation of the authors' book in the German language, it developed into an entirely new book.

In one volume the authors have demonstrated the happy faculty of combining the clinical and the experimental phases of each subject discussed. The one almost imperceptibly runs into the other, erasing the artificial division which has hitherto been maintained. For example, the subjects of the auditory and vestibular functions of the ear are discussed clinically in all their phases and, without the reader's perceiving it, the experimental work on which the clinical facts are firmly based is introduced as if it were one and the same subject, which it ought to be.

Ordinarily to the clinician the experimental work on human beings as well as on animals is a thing apart from his daily clinical thinking. To him experiments are products of experimental laboratories represented by articles in journals written by laboratory workers in a technical and highly complicated fashion which is too difficult and too dull to follow. Besides, the articles are dispersed in various journals with which the clinician is hardly acquainted. The authors of this book bring all the work together in conjunction with the clinical data and, as stated, make it seem amazingly like one subject.

Seventy pages of references refer to the original sources of the knowledge of the topics discussed. These include the important European and American contributions and are sufficiently and clearly enough abstracted in the text that one need not refer to the sources unless further details of a certain topic are desired. Much of the original experimental work done by the authors in the course of many years is included in the book, together with many explanatory diagrams and actual photographs.

All the complicated conditions which confront the ophthalmologist and the otolaryngologist, such as abscess of the brain and other intracranial complications of diseases of the eye, of the ear and the nose, are clearly presented and their neurologic applications correlated.

The authors by bringing forth this publication have made a distinct contribution to the English literature on the subjects included in their survey.

After a glance through the pages of this book, it would be hard to conceive why an ophthalmologist or an otolaryngologist would want to be without it.

The Trials and Triumphs of the Surgeon and Other Literary Gems. By J. Chalmers Da Costa. Edited by Frederick E. Keller, M.D. Price $5. Pp. 401. Philadelphia: Dorrance & Company, Inc., 1944.

The collected works of Dr. J. Chalmers Da Costa are presented in a fine book of 401 pages. The following interesting subjects are discussed: "The Antivivisection Question," "The History of Medicine," "The Alexandrian School," "John Johnston," "Some Figures of the Past," "A Glimpse of the London of the Restoration," "Objectionable People and Their Unjustifiable Longevity," "Francis X. Dercum," "George McClellan," "Birthday Celebration Address in Honor of Dr. W. W. Keen," "Stepping Stones and Stumbling Blocks," "Some Surgical Fashions and Customs," "In the Days of Polypharmacy," "Something About Medical Ethics," "Duties of a Hospital," "Address Presenting Memorial Tablet to Jefferson Medical College," "The Bugle," "Address at Commencement Exercises of Woman's Medical College," "The Resident Physician—Address Before Philadelphia County Medical Society" and "Address on the Occasion of the Graduation Exercises at the Naval Medical School in Washington on March 30, 1907."

This book, though not devoted to otolaryngology, merits high praise in this review because it reveals the spirited literary power and varied knowledge of the distinguished Dr. Da Costa. It should be in the library of every physician. The proverbs, the anecdotes and the vivid historical accounts throughout the chapters make for interesting reading whether in one's study or on a vacation.

It is edited by Dr. Frederick E. Keller, one time student of Dr. Da Costa, a surgeon in his own right and a man of letters, who has brought this particular selection together for the first time. He has conducted extensive researches on the life of his mentor, and in this book one sees Dr. Da Costa not only as a brilliant surgeon, teacher and analytic scholar but as a poignant wit and a noble character.

He becomes alive again to those who knew and loved him and is refreshingly introduced to those not privileged to have known him.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.

Place: Philadelphia. Time: June 18-22, 1945.

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President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.

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President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland.

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President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.

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American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.

Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

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Eastern.—Chairman: Dr. Oram R. Kline, 414 Cooper St., Camden, N. J.

Southern.—Chairman: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.

Middle.—Chairman: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis.

Western.—Chairman: Dr. Aubrey G. Rawlins, 384 Post St., San Francisco.

American Otological Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.

Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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